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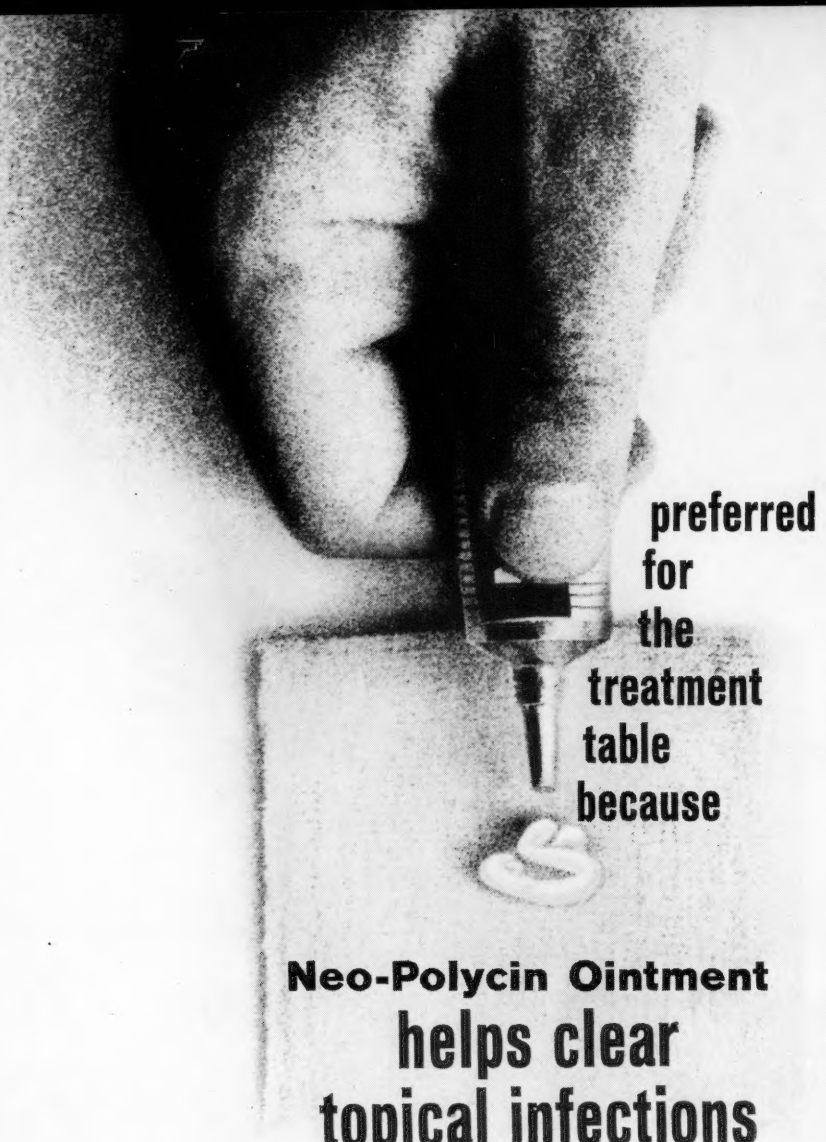

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The Industrial Medical Association was organized in 1915 as the American Association of Industrial Physicians and Surgeons. Its first annual meeting was held the following year. In 1951 the present name was adopted.

The purposes of the organization as stated in the certificate of incorporation (not-for-profit) are:

"The object of this Association will be to foster the study and discussion of the problems peculiar to industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes of the medical care for employees; and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for their mutual advancement in the practice of their profession."

Presently, the membership includes more than 4000 physicians in the United States, Canada, Latin America, and other parts of the world. Physicians "who have an interest in Industrial Medicine" may apply for membership; specialization in this field is not essential. Regionally, the Industrial Medicine Association has 15 Component Societies in the United States. The majority of the Association's members carry their memberships through one of the component groups.

The Journal of Occupational Medicine, owned and published by the Industrial Medical Association, is sent monthly to each member as one of the advantages of membership.

The annual business meeting and convention is held in the spring. Since 1945, the convention has been held concurrently with the meetings of other organizations having related interests including the American Association of Industrial Nurses, Inc., and the American Association of Industrial Dentists. This combination of meetings is known as the Industrial Health Conference.

The Occupational Health Institute, Inc., is a non-profit educational organization created by the Industrial Medical Association to promote the cause of health in industry. Since 1951, the Occupational Health Institute has conducted the program of evaluation, approval and certification of Medical Services in Industry, on the basis of standards established by the Standards Committee of the Industrial Medical Association. This program was conducted by the American College of Surgeons for many years until its transfer to the Association.

The Association's headquarters office serves as a focal point for the Association's activities and through its coordination of information regarding industrial health problems encountered throughout the country, it offers invaluable assistance to members on a host of specific subjects.

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Biltmore Hotel, Los Angeles, Calif. **April 11, 12 and 13, 1961**

Journal of Occupational Medicine

Occupational Health Service Experiences of Two Hotels for 1955 and 1956

Josephine A. Cipolla, R.N., M.P.H., J. W. Meigs, M.D., and M. L. Brown, M.A., New Haven, Conn.

THIS STUDY reports the 1955 and 1956 industrial accident experience of two hotels: Hotel A, which provided an occupational health service for its 876 employees; and Hotel B, which did not provide this type of service for its 357 employees.

Information is not available as to how many of the 14,000 hotels in the United States have occupational health services for their employees, but it is believed that only very large hotels provide such a program. Hotels range in size from 10 to 3,000 rooms.[1] Only 280 of the 14,000 hotels have more than 500 rooms. It is estimated that a hotel with 500 rooms would employ between 300 and 400 persons and it is probable that very few smaller hotels provide occupational health services at present.

Method of Investigation

The information for each compensable accident in Hotels A and B for the years 1955 and 1956 was transferred directly from hotel and insurance company records onto a work sheet. To eliminate as many variables as possible, certain groupings were made. All information was classified in the direct transfer of the data from the records to the work sheets.

The injuries sustained by workers at their place of employment in New York state, which is the site of Hotel A, are classified by the Workmen's Compensation Law into two types:

These classifications were used at Hotel A for the years 1955 and 1956.

1. *Nonreportable injury.* This is a minor injury whose total treatment requirements are a maximum of two first-aid nursing treatments. It is not reported to the Workmen's Compensation Board.

2. *Reportable injury.* This is an injury which needs medical treatment in place of and/or beyond the two first-aid nursing treatments. It is reportable to the Workmen's Compensation Board.

The industrial injuries in the state of Connecticut, site of Hotel B, are not classified by law in the same categories as are those of New York state. The Connecticut employer may provide a physician of his choice to treat his injured workers. The physician may be an employee of the industry, or his services may be contracted on a fee-for-service basis. In either case, he may provide medical direction to a nurse in the industrial establishment. The nurse may render care for any industrial injury according to her professional judgment. This is the only method for limiting the number of nursing treatments for an industrial injury.

Only an industrial injury which causes one or more days of disability or is by definition disabling (e.g., amputation of fingertip) is reportable to the Connecticut Workmen's Compensation Commissioner. Since the physicians who treated the industrial injuries of Hotel B practiced on a fee-for-service basis, all injuries which they treated had to be reported to the

From the Section of Occupational Health, Department of Public Health, Yale University.

This article is based on a thesis presented by the senior author to the Department of Public Health, Yale University, in partial fulfillment of the requirements for the degree of Master of Public Health, 1958.

Submitted for publication March 7, 1959.

insurance company in order for them to collect their fees. Injuries involving indemnity or other expenses were also reported to the compensation insurance carrier. For this study, the injuries of Hotel B were classified only as reportable injuries, as follows:

Reportable. This included all injuries for which the insurance company had records. These included injuries for which there were medical, and/or indemnity and other related expenses.

There were no nonreportable injuries for Hotel B because records were incomplete.

The number of days of disability for each accidental injury was obtained from the medical records of Hotel A. Records that were doubtful were verified at the office of the insurance carrier. The total days of disability for employees at both hotels include the disability waiting period specified by the Connecticut and New York State Compensation Laws. The waiting period of seven days was included, even though the employee is not compensated for it except as regulated by law.

The medical and associated costs of the compensation (reportable) cases were obtained from the records of the insurance carrier. Medical costs include physicians' fees, hospital fees, ambulance fees, medical equipment and supply charges, and all other direct-cost items such as braces and back supports, laboratory fees, and private duty nurse fees. Certain additional costs are referred to by the insurance carriers as "expenses." These are fees for legal services and for medical consultations which may be requested by the carrier. Compensation costs include compensation payments for disability based on a percentage of the employee's wages, and awards for permanent disabilities and for death. They also include the costs of those cases closed by stipulation.

Results

Table I shows that in 1955 the workers of Hotel A experienced 181 injuries as compared to 154 injuries in 1956. The figures represent a reportable-injury frequency rate of 90 in 1955 and 84.8 in 1956. In Hotel A, comparable methods of recording existed during each year, the accident prevention program was uniform during the two-year period, and the professional staff in the occupational health department remained the same.

The employees of Hotel B experienced 94 "reportable" injuries in 1955 and 58 "reportable" injuries in 1956. The "reportable"-injury frequency rates for Hotel B were 101.8 in 1955 and 65.4 in 1956. Neither the numbers nor the rates are considered to be reliable because of a change in administrative reporting policies from year to year. A review of the records during this study showed that in 1955 management was notified by workers of 12 injuries for which the insurance company had no record. In 1956, there were 27 injuries reported to management for which the insurance company had no report. If these were added to the "reportable" injuries in their respective years, the totals for Hotel B would be more nearly equal for both years. But since these injuries were not reported to the insurance company, they could not be studied adequately and were excluded from the annual total numbers of "reportable" injuries for Hotel B.

The reportable-injury frequency rates in the two hotels cannot be reliably compared for the reason given above and for another related to reporting. The workers of Hotel A, where a nurse was employed, were more likely to report their injuries than the workers of Hotel B, which did not provide nursing service. This is indicated by the fact that there were more than 200 nonreportable injuries treated by the nurse in each year in Hotel A. The occupational hazards in the two hotels were basically similar and there were probably equivalent numbers of minor injuries in Hotel B, but the absence of detailed data makes further speculation un-

Table I—Reportable-Injury Frequency Rates, Hotels A and B, 1955 and 1956

Year	Total Number of Injuries	Number of Man-Hours Worked	Average Number of Employees	Frequency Rate*
		Hotel A		
1955	181	1,911,144	876	90.0
1956	154	1,815,368	850	84.8
		Hotel B		
1955	(94)	923,016	357	(101.8)
1956	(58)	888,264	337	(65.4)

*American Standard Method of Recording and Measuring Work Injury Experience, American Standards Association, New York, 1954, p. 8.

Table II—Number of Reportable Injuries, Nonreportable Injuries, and Number of Injuries per 100 Employees, Hotel A, 1955 and 1956.

	Hotel A	
	1955	1956
Number of employees	876	850
Number of injuries		
Reportable	181	154
Nonreportable	208	203
Number of injuries per 100 employees	44.7	42.1
Reportable	20.7	18.1
Nonreportable	24.0	24.0

Table III—Disabling-Injury Frequency Rates, Hotels A and B, 1955 and 1956

Year	Number of Disabling Injuries	Number of Man-Hours Worked	Average Number of Employees	Disabling Injury Frequency Rate*
		Hotel A		
1955	25	1,911,144	876	13.1
1956	18	1,815,368	850	9.9
		Hotel B		
1955	11	923,016	357	11.9
1956	9	888,264	337	10.1

*American Standard Method of Recording and Measuring Work Injury Experience, American Standards Association, New York, 1954, p. 8.

Table IV—Disabling-Injury Frequency Rates, Hotels A and B, Hotel Industry, State of Connecticut and of the United States, 1955 and 1956

	1955	1956
Hotel A (N.Y.)	13.1	9.9
Hotel B (Conn.)	11.9	10.1
Hotel Industry, State of Connecticut*	9.1	7.1
Hotel Industry, United States†	12.6	11.9

*_Safety Views and News_, Connecticut Labor Department, IX: 1, (Dec.) 1957, p. 9.

†News from the U. S. Department of Labor, Injury Rates by Industry, 1956, Bureau of Statistics, Washington, D.C., U.S. Government Printing Office Dec. 5, 1957, p. 7.

desirable. Because of the probable unreliability of numbers of reportable injuries for Hotel B, these numbers and the rates derived directly from them are put in parentheses in Table I.

Table II shows that in Hotel A the numbers of injuries per 100 employees per year remained about the same in the two years in both reportable and nonreportable categories, with slightly more than half of the total in the nonreportable group. Results for Hotel B were similiar insofar as data were available, with 26 reportable injuries per 100 employees per year in 1955 and 17 in 1956. They are not presented in tabular form because of the previously mentioned questions of reliability.

Table III shows that disabling-injury frequency rates ranged between about 10 and 13 over the two-year period, with the 1956 figure slightly lower than that for 1955 in each hotel. For Hotel B, the disabling-injury frequency rate may be a more reliable basis for comparison of injury experience with Hotel A because disabling injuries were defined and recorded in a more nearly uniform manner in the two hotels.

Table IV shows the disabling-injury frequency rates of Hotels A and B and the rates in the hotel industry in the state of Connecticut and the United States for the years 1955 and 1956. The disabling-injury frequency rates for the hotel industry in the state of New York in which Hotel A is located are not available. A comparison of the rates of Hotel A with the rates of the United States shows that there was no significant difference between the two either in 1955 or 1956. For Hotel B, the disabling-injury frequency rate was somewhat higher than the Connecticut rate and slightly lower than the United States rate for both years. This difference is not statistically significant.

Table V shows that Hotel A had a lower severity rate than Hotel B for each year of the study. The rate for Hotel A dropped by about

Table V—Severity Rates, Hotels A and B 1955 and 1956

Hotel	Year	No. of Lost Days	No. of Man-Hours	Severity Rate*
A	1955	695	1,911,144	364.0
	1956	437	1,815,368	241.0
B	1955	410	923,016	444.0
	1956	409	888,264	460.0

*American Standard Method of Recording and Measuring Work Injury Experience, American Standards Association, New York, 1954, p. 10.

Table VI—Numbers of Disabling Injuries and Average Number of Lost-Time Days per Disabling Injury, Hotels A and B, 1955 and 1956

Hotel	Year	Number of Disabling Injuries	Average No. of Days Lost per Disabling Injury*
A	1955	25	27.8
	1956	18	24.3
B	1955	11	37.3
	1956	9	45.4

*American Standard Method of Recording and Measuring Work Injury Experience, American Standards Association, New York 1954, p. 10.

Table VII—Numbers of Employees, Man-Hours, per Employee Hotels A and B, 1955 and 1956

	Year	Total No. of Employees	Total Man-Hours Worked	Man-Hours per Employee
A	1955	876	1,911,144	2,182
	1956	850	1,815,368	2,136
B	1955	357	923,016	2,583
	1956	337	888,264	2,636

Table VIII—Compensation Costs per Employee, Hotels A and B, and Occupational Health Service Costs per Employee Health A, 1955 and 1956.

	Year	No. of Employees	Costs Compensation Total	Per Emp.	Costs Occupational Health Total	Per Emp.	Total Costs Total	Per Emp.
Hotel A	1955	875	$14,075	$16.07	$8,894	$10.15	$22,969	$26.22
Hotel B	1955	357	$14,557	$40.50	None	None	$14,557	$40.50
Hotel A	1956	850	$ 8,989	$10.57	$9,073	$10.67	$18,602	$21.24
Hotel B	1956	337	$11,384	$33.78	None	None	$11,384	$33.78

one-third in 1956, while the severity rate for Hotel B rose slightly in 1956.

Table VI shows that the average number of days lost per disabling injury varied between 24.3 and 45.4. The rate for Hotel A was somewhat lower than that for Hotel B.

Table VII shows differences in the average number of man-hours worked per employee in the two hotels. Whereas Hotel A was stable at about 2,100 hours per employee per year, or about 42 hours per employee per week, Hotel B had considerable overtime, with a 52-hour week average in 1955 and 53 hours per employee per week in 1956. No adjustment has been made in these computations for the "extra" employees who are not counted in the total numbers of employees, although their man-hours worked are included in the total man-hours. It is known, however, that the number of workers in this "extra" category would not affect these results significantly.

Table VIII provides a comparison of costs of health services in the two hotels. The problems related to comparing the data on the basis of the numbers of the reported injured of both hotels were described earlier. Under these circumstances, a more accurate basis for comparison of costs is the total number of employees instead of the number of injuries.

For both hotels, the term "compensation costs" included all medical costs of occupational cases whether or not compensation payments were made. These medical costs include payments to physicians inside or outside the hotel, as well as all other direct treatment costs. For Hotel A, the direct costs for all reportable-injury cases were included in compensation costs. Total costs for Hotel A included occupational health service costs, specifically, (1) rental value of the office space; (2) cost of medicine, equipment, and supplies; (3) medical fees for physical examinations of applicants and employees; and (4) nurse's salary. This information was provided by the management or taken from health service records.

Compensation costs for both hotels also included weekly or lump-sum compensation payments as required under the Workmen's Compensation Acts and by administrative decisions.

Table VIII shows a decrease from 1955 to 1956 in compensation costs per employee in each hotel. But the per capita compensation costs of Hotel B were from two to three times those of Hotel A for each year. When the occupational health service costs at Hotel A were added to compensation costs, total costs at Hotel B were still 50% greater than those of Hotel A. Hotel B, in Connecticut, operated under a different compensation act than Hotel A, in New York. The Connecticut Act provided slightly higher weekly benefits ($40.00 vs $36.00) and had no published fee schedule. It was not possible to make a direct comparison of medical fees and costs for comparable injuries. From over-all estimates, the physicians' costs per case in Connecticut were probably about 50% higher than in New York. This did not account for more than a part of the increased compensation costs in Hotel B. The major portion of Hotel B's compensation costs was from indemnity payments, including lump-sum settlements, as presented in Table X.

Taken as a whole, Table VIII suggests that the occupational health service at Hotel A provided an important factor in controlling costs, namely, continuity of care. It also provided accurate records on which to base cost analyses. For example, 83% of reportable injuries at Hotel A in the two years were handled by the occupational physician. He saw each employee an average of 5.4 times, and the average cost of each visit was $3.94. In contrast, at Hotel B only 22% of reportable injuries could be analyzed in detail, and the analyses were not comparable with Hotel A because of incompleteness of the sample. Consequently, only the over-all data, known to be reasonably reliable, have been presented in the tables.

The annual costs of the occupational health services for employees in Hotel A averaged $10.41 per employee. Hotel B did not provide these services. Hotel B paid more compensation costs per employee than Hotel A paid for combined compensation and occupational health services cost per employee, besides which Hotel B employees received none of the preventive health services provided for the employees of

Hotel A. For $10.41 per employee per year, Hotel A workers received all the services of an occupational health program, including treatment of nonreportable injuries and the preventive services. Actually, the cost of employee health service was less than $10.41 per worker. This is true because the facilities and part of the nursing time were used for guest service, and, in addition, a small proportion of nursing time was used for assisting the doctor during the care of his private patients as indicated in Table IX.

Table IX indicates the workload of the occupational health service of Hotel A. When the data were available, the number of visits was shown in preference to the number of cases. These figures suggest the relative amounts of time which the nurse utilized for her various functions.

Table X shows that on the basis of the average indemnity cost per disabling injury, Hotel B paid three times as much as Hotel A in 1955 and more than five times as much as Hotel A in 1956. Hotel A experienced a substantial decrease in average indemnity cost per disabling injury in 1956, while the average indemnity cost per disabling injury of Hotel B decreased only slightly in 1956. When compared with Hotel A's costs, the indemnity costs of Hotel B were greater than would be expected from either lost-time injury or severity rates. This difference may be due to the fact that Hotel B closed six cases by stipulation in both 1955 and 1956.

In studying the times of day in which the injuries occurred, an answer was sought to the question of whether or not accidents had a greater tendency to occur during certain periods of the day. The hours were grouped to correspond as much as possible to working shifts and to busier periods of hotel operations, such as meal service hours and checkout time. The complexity of hotel operations made these objectives difficult to obtain.

The data show that about 33% of the reportable injuries in each hotel occurred between 1:00 p.m. and 4:00 p.m. At Hotel A, an additional 25% occurred between 10:00 a.m. and 1:00 p.m. At Hotel B, an additional 20% occurred between 7:00 p.m. and 10:00 p.m. There was no detectable distribution pattern for the remaining injuries in either hotel.

Accident causes were studied for each hotel, and data for the two years were combined. The commonest cause was the worker's being struck by an object or striking against an object, for example utensils, cutlery, equipment, and hand trucks. These miscellaneous objects caused about 33% of injuries. "Working surfaces," such as slippery floors, were the causal factor in about 17% of injuries, while the position of the worker, for example, reaching, bending, lifting, and stooping, caused 12%. Hot substances or equipment were responsible for about 10% of injuries, and the remaining causes were scattered. At Hotel B, 22% of the causes were not recorded in contrast to Hotel A's 3% not recorded.

Accident type describes the manner in which the agency produced the injury. If the totals in each category are grouped together for the

Table IX—Care Provided by the Occupational Health Department Personnel Hotel A, 1955 and 1956

Number of Cases or Visits as Noted*	1955	1956
Reportable occupational injuries or illnesses (Visits)	798	704
Nonreportable occupational injuries or illnesses (Visits)	246	238
Return-to-work interviews (cases)	181	84
Nonoccupational injuries, illness, problems (visits)	719	533
Medical examinations (cases)	720	830
Guest-patients (cases)	579	442
Private patients (visits)†	300	300
Total	3,545	3,131

*Figures are from complete records except where indicated.

†Estimated.

Table X—The Average Indemnity Cost per Disabling Injury Hotels A and B, 1955 and 1956

Hotel	Year	Number of Disabling Injuries	Average Indemnity per Disabling Injury
A	1955	25	$264.41
	1956	18	$142.90
B	1955	11	$811.64
	1956	9	$771.24

Table XI—Number of Reportable Injuries According to Accident Types Hotels A and B for the Combined years 1955 and 1956.

Accident Types	Hotel A	Hotel B	Hotels A & B
Struck by	89	29	118
Striking against	72	25	97
Slip (not fall) or overexertion	57	19	76
Exposure to temperature extremes	38	13	51
Fall on same level	30	7	37
Inhalation, absorption, ingestion	22	3	25
Caught in, by, or between	10	12	22
Fall to different level	4	2	6
Continuous occupational activity	2	2	4
Other accident types	2	4	6
Unknown	9	36	45
Totals	335	152	487

Table XII—Number of reportable Injuries According to the Nature of the Injury, Hotels A and B, for the Combined Years, 1955 and 1956

Nature of the Injury	Hotel A	Hotel B	Hotels A & B
Contusions	98	29	127
Lacerations	65	36	101
Sprains, strains	68	23	91
Second, third-degree burns	39	4	43
Fractures, dislocations	15	5	20
Dermatitis	13	3	16
Imbedded foreign bodies	10	0	10
First-Degree burns	2	8	10
Paronychia	7	1	8
Abrasions	7	0	7
Loose foreign bodies (Eye)	5	2	7
Hernias	2	0	2
Other	1	6	7
Unknown	3	35	38
Totals	335	152	487

Table XIII—Number of Nonreportable Injuries According to the Number of Nursing Treatments per Injury, Hotel A, 1955 and 1956

	1955	1956
Total number of non-reportable injuries	208	203
Number of nursing treatments per injury		
One nursing treatment	170	168
Two nursing treatments	38	35

two-year period for each hotel, the following patterns emerge. Accidents in which workers were struck by something accounted for the greatest number of injuries. These injuries may have been lacerations, contusions, burns, fractures, foreign bodies, or abrasions. The accident type which caused the second greatest proportion of injuries aws also the same for both hotels. This was the type in which workers struck against something, generally producing less severe injuries than the first type. The third most common type for both hotels were accidents in which the workers slipped (not fell) or overexerted themselves. The more common results were strains and sprains. The fourth most common type of accident in each hotel was exposure to temperature extremes, invariably causing burns.

The data show that the injuries occurred in similar locations upon workers at both hotels. About 25% of the injuries in each hotel affected the right hand or wrist. The left hand or wrist was the next most common site of injury.

The nature of the injuries were also comparable. Contusions, sprains or strains, and lacerations accounted for about 63% of the injuries at both hotels.

The nature of the injury classified as "other" for Hotel A was an irritation of an eye. The six injuries classified as "other" for Hotel B were bleeding (vaginal), nosebleed plus fainting, numbness of the hand, two eye irritations, and one case of electric shock.

Infections occured in both hotels. Hotel A workers experienced eight infected wounds during the two-year period while Hotel B workers experienced two infections.

Table XIII shows how nonreportable injuries were handled by the nurse at Hotel A in both years. This care was administered by the nurse within the first-aid-treatment requirements as defined by law, but it was given under the medical direction of a part-time physician.

The majority of these nonreportable injuries required only one nursing treatment. Approximately one in five injuries required two nursing treatments.

The 181 reportable injuries sustained by the workers of Hotel A in 1955 were treated by the doctor. Approximately 50 of these injured workers, however, were given their first treatment by the nurse and were not seen by the doctor during this initial treatment. Of these 50 workers, six received second treatments by the nurse before they were examined by the physician. In 1956 in Hotel A, the workers sustained 154 reportable injuries. Before 61 of these workers were treated by the doctor, they received one treatment by the nurse. Of these 61 workers, two received second treatments by the nurse before they were treated by the doctor.

Conclusions

1. There are measurable differences between the industrial accidental-injury experience of Hotel A, which provided an occupational health program, and Hotel B, which did not provide this service for its employees. Hotel A had (1) lower disabling-injury severity rates; (2) fewer number of lost-time days per disabling injury; (3) fewer cases whose compensability was doubtful or disputed; (4) more complete records of the professional care which was provided to injured workers; (5) lower average medical-indemnity and expense payments per employee per year; and (6) a more nearly accurate picture of accident experiences upon which to formulate a safety and health program.

2. The analysis of the industrial-accident experiences of Hotel A and Hotel B showed similarities as follows: (1) reportable-injury frequency rates; (2) disabling-injury frequency rates; (3) patterns of reportable accident by agencies: miscellaneous objects; working surfaces; position of worker; and hot equipment

or substance; (4) patterns of reportable injuries by types: struck by; struck against; slip (not fall) or overexertion; and exposure to temperature extremes; (5) patterns of locations of reportable injuries: the right hand or wrist; the left hand or wrist; foot or ankle; and lower back; and (6) patterns of reportable injuries according to nature of injury: contusions; lacerations; and strains and sprains.

3. Hotel A had more nonreportable than reportable injuries in each of the two years. Since the data on reportable injuries suggest that accident experience was similar in the two hotels, it seems probable that Hotel B also had a considerable number of nonreportable injuries. The favorable data on severity of injuries, total cost, and total health service experience at Hotel A support the view that it is desirable to find and treat as many minor injuries as possible. Hotel A's experience further suggests that the professional nurse working with a part-time physician can safely and completely treat more injuries than she needs to refer to the physician.

4. State Compensation Laws affect costs. In the state where medical fees were not scheduled and there was no occupational health service, the costs of medical care were higher.

5. When professional persons were responsible for the followup of injured workers, there was less lost time and lower medical and indemnity costs.

6. A qualified occupational health nurse, working with a part-time doctor, provided care at the place of employment for workers with industrial injuries in addition to carrying on a preventive program.

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Medical Care and Welfare Program for Hotel Workers in New York City

Frank P. Guidotti, M.D., New York, N.Y.

THE HOTEL industry medical care and welfare plan is an example of one of the larger labor-management plans instituted through collective bargaining. The parties to the bargaining are the Hotel Association of New York City, Inc., and the New York Hotel Trades Council, AFL-CIO. The Hotel Association represents most of the larger hotels in New York City. The Hotel Trades Council is a council of 10 affiliated local unions and represents approximately 34,000 employees working in 185 union contract hotels.

The 1944 collective bargaining agreement was the first to provide for a welfare program in a hotel industry. That agreement called for employer contributions at 3% of the payroll to finance various forms of insurance for employees and their families and today has been increased to 3.25%, amounting to a little more than $3,000,000 a year deposited in a trust fund governed jointly by a board of trustees and an advisory committee, representing both labor and management.

Benefit payments under the plan started March 1, 1945, and included life insurance, accidental death and dismemberment insurance, weekly accident and sickness insurance, and Blue Cross hospitalization insurance. The last-named applied to the family as well as to the individual member.

Soon after the welfare program was inaugurated in 1945 it became apparent that the great majority of hotel workers were receving inadequate medical care—inadequate when compared with that of the community at large. Experience with the accident and sickness program showed that a large part of its utilization was by those who had previously obtained medical care from "outpatient" departments of municipal hospitals, and that "inpatient" care was too frequently the result of emergency hospitalization. After four years of study and collective bargaining, the parties to the agreement established a jointly administrated health center to furnish free comprehensive ambulatory care and "in-hospital" medical and surgical care to 34,000 unionized employees in the industry, the cost of which is borne exclusively by management.

A five-story building, with 25,000 square feet of space, was purchased, renovated, and equipped to provide the needed services. To safeguard the continued operation of the program and the quality of medical care it would furnish, a special act of legislation was passed by New York State in 1949 authorizing the creation of the health center as a membership, nonstock corporation, under the supervision of the New York State Department of Social Welfare, to provide comprehensive ambulatory medical care through a self-owned and operated health center, and "in-hospital" medical and surgical care.

In 1954 the welfare program was further extended to include a pension plan to which the employers contribute an additional 2% of the payroll, amounting to approximately $2,000,000 a year. At the present time there are 2,045 retired workers eligible for medical care at the health center. All of these retired workers are receiving pensions.

As of June 1, 1957, through additional collective bargaining agreements, employers contribute $1.00 per week (2½ cents per hour) per employee for the inclusion of families in the medical care program. This contribution amounts to approximately $1,600,000 per year. The plan, still under study, will commence in the near future and will include 47,000 de-

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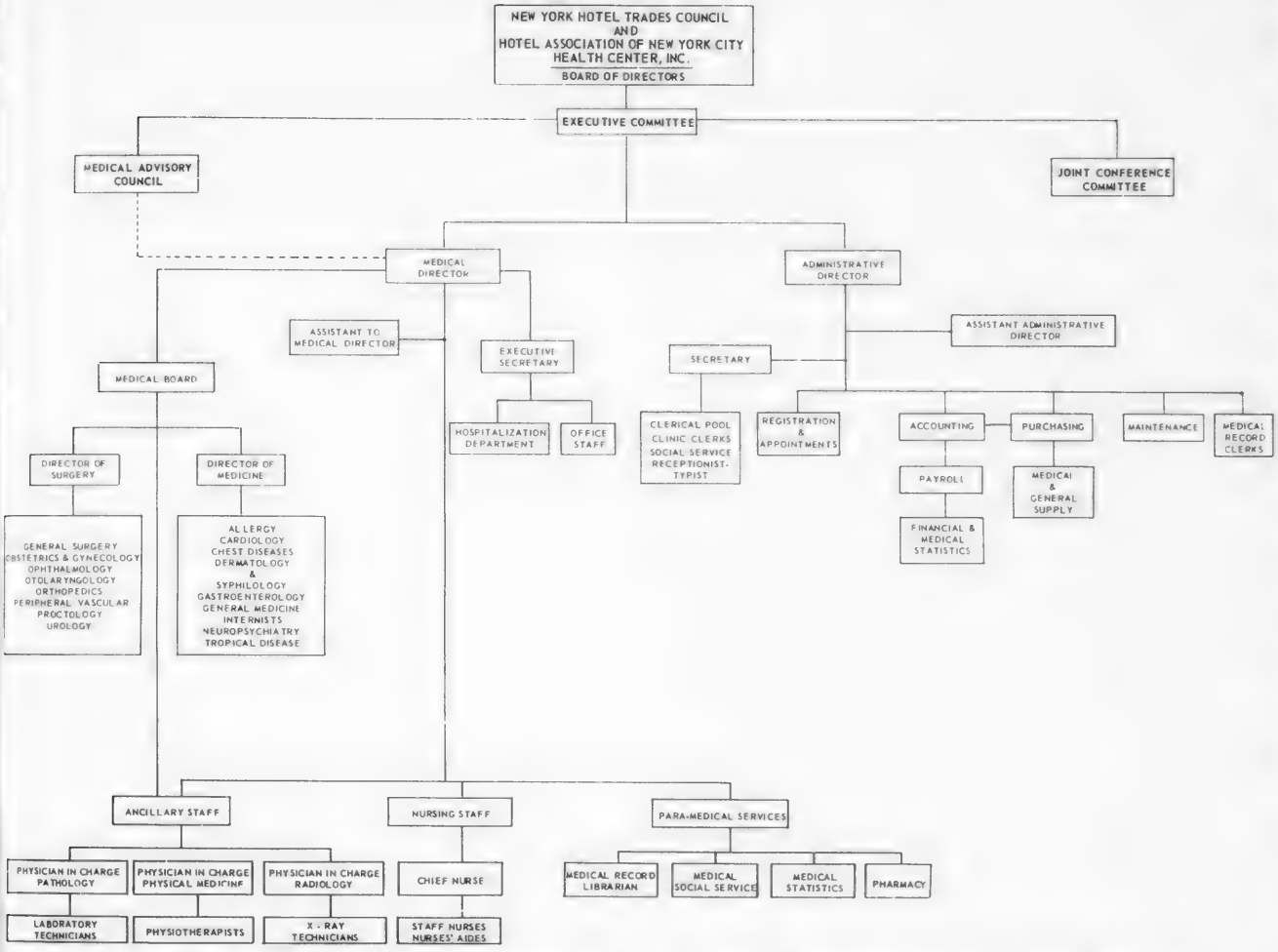

pendents, bringing the total coverage to 80,000 individuals for comprehensive medical care.

Employer contribution for the entire welfare program is approximately $7,000,000 per year.

Organization and Administration

The health center is governed by a board of directors, composed of 20 members—10 representatives from the Hotel Trades Council and 10 from the Hotel Association of New York City, Inc.—who control all affairs and business of the corporation (see the chart). The medical director and the administrative director are ex-officio members of the board of directors; the medical director participates in all policy decisions which may directly or indirectly influence the medical service rendered, and the administrative director participates in policies affecting the business management of the health center.

Prior to the beginning of the program on Oct. 25, 1950, a medical advisory council was appointed. At the present time this council comprises 16 members—15 leading physicians in the New York area, well known in the allied fields of public health, occupational health, industrial hygiene; and the medical director. Included in the membership and representing the medical staff are the director of surgery, the director of medicine, and the chief of one of the subspecialty services.

Functions of the medical advisory council are to promulgate regulations pertaining to professional and technical services, to examine the qualifications of physicians recommended for appointment, and to guide the board of directors in the formulation of professional policies.

The medical director is ably assisted by the medical board, which is composed of 16 members of the medical staff—the director of surgery, the director of medicine, and the chiefs or physicians in charge of all specialty and subspecialty clinics. It is the function of the

medical board to supervise the professional conduct of the medical staff in all clinics and to resolve any problems that may arise in the operation of the health center.

Medical Staff

The present medical staff includes 23 specialist consultants, 86 physicians in surgical services, and 81 physicians in medical services—totaling 190 physicians. Eighty of this total are on active clinic duty; the remainder serve as alternates for clinic duty, for emergency house calls, and as members of the hospitalization staff. During the past year 146 members of the medical staff participated actively in our medical care program, and 58 nonstaff physicians participated in rendering emergency medical care and hospitalization.

Professional Staff Qualifications

Of the 160 specialists on the medical staff. 145 (90%) are diplomates of specialty boards and 15 are nondiplomates (several have passed the first part of their board examinations and others are Fellows of the American College of Physicians and Surgeons). Affilation of the specialists with accredited hospitals is as follows:

Consultants	5
Directors of medical surgical services	25
Assistant directors, medical services	5
Chiefs of medical services	16
Full attending	38
Associate attending	42
Assistant attending	24
Clinical assistants	5
	160

Ninety-five (60%) have teaching affiliations with medical and postgraduate medical schools as professors and instructors in the various grades. Administrative personnel number 37, ancillary medical technical staff 28, and 5 on the maintenance staff.

Extent of Medical Care

Comprehensive diagnostic, ancillary, and therapeutic ambulatory care provided in the health center focuses in the general medicine clinic, which serves in two most important capacities: (1) providing the patient with the equivalent of a "family physician" and (2) evaluating and screening of medical findings prior to the referral of a patient to specialty clinics for consultation and care if needed. Morning and afternoon general medicine clinic sessions are held daily. Each session is staffed by five well-qualified general practitioners and supervised by a specialist in internal medicine, serving as medical consultant.

The emergency clinic provides first-aid treatment by a general practitioner and evaluates immediate need for referral to specialty clinics for consultation or care *prior* to referral to the general medicine clinic.

The active staff of the general medicine clinic is composed of 17 general physicians and 6 internists. All general physicians have at least an appointment in an accredited hospital as a clinic assistant. Each clinic physician sees an average of three patients per hour, affording adequate time for each patient. The majority of these physicians are on duty daily, thus affording better assurance of continuity of care by the same general physician.

On the patient's first visit routine laboratory tests are performed: hemoglobin, urinalysis, serology (to rule out syphilis), and a minature chest x-ray. An appointment is made for a visit to the general medicine clinic, at which time a thorough physical examination is made, and specialty clinic referrals, medical consultation, and/or further diagnostic tests are ordered if required. Thus, prevention and treatment of disease are equally emphasized.

Patient Services at the Health Center

A total of 83,593 visits to physicians were made by 11,927 individual patients to general medicine and specialty clinics during the health center's eighth year of operation, i.e., an average of seven visits per patient. The current figure represents the greatest number of physician services furnished in any year. In conjunction with these visits to physicians, 57,232 diagnostic procedures were performed, and 19,815 ancillary therapeutic services were furnished to patients. Medical care by physicians was divided almost equally between the specialty and general medicine services.

A significant number of medical services on a nonappointment basis were given in the emergency clinic, which is the major point of entry for new patients, and most of the patients introduced to the center through this clinic return for complete medical examinations and continuing medical care.

Special medical services are offered on a voluntary basis. These include (1) preplacement examinations; (2) periodic examinations; (3) return-to-work examinations following illness; (4) first aid for occupational injuries; (5) usual immunization program; (6) routine electrocardiography for all employees 45 years of age and over; (7) routine stool examinations; (8) prenatal care; (9) diagnostic services for private or family physicians of hotel workers, including reports and abstracts of pre-

vious treatment, when requested and authorized by the worker; (10) rehabilitation; (11) advice on social service problems; and an (12) educational program.

Medical Follow-Up

Follow-up services are valuable to both patient and physician in determining the effectiveness of the ambulant care and are one of the important functions of the medical record department in its daily review of all charts returned from clinics, assisted by the medical social service department in contacting the patient if pathology findings indicate need for immediate care.

In 1957 a program was initiated which we believe is unique in outpatient clinic care. Voluminous and complicated charts are reviewed by one of our internists at periodic intervals and a short but complete summary of the patient's medical history is placed in the front of the chart. This not only saves the physician time, but also calls his attention to significant factors in the past history of the patient. In certain instances, it has led to the discovery and correction of oversights and omissions.

Medical Services Outside the Health Center

Medical care provided outside the health center includes in-hospital medical, surgical, and maternity care, ambulance service, visiting nurse service, emergency house calls for first-aid treatment and/or to determine need for hospitalization, and visits by appointment to the offices of members of the specialist consultant staff for diagnostic services and treatment, such as (1) radioactive iodine study and therapy; (2) neurosurgical consultation (3) endoscopy; (4) encephalography; and (5) deep x-ray therapy for benign conditions.

Special Problems

Several special problems of the hotel industry have warranted special clinics for study and care, one of these being tropical disease. The present Puerto Rican population of New York City is approximately one-half million, and 20% of health center patients are of Puerto Rican origin. Since September, 1953, all food handlers in the hotel industry and all workers who have migrated from tropical areas within the previous five years have had routine stool examinations for parasities. Of particular importance from the public health standpoint was the relatively high incidence of amebiasis, and from this standpoint alone it is felt that this study of tropical disease has been valuable.

Physician' Services and Costs

Payments to Physicians in the Health Center

Two internists, seven general practitioners, and one roentgenologist serve on a half-time basis and receive an annual salary and all fringe benefits. The other 10 general practitioners work three-hour sessions, averaging nine hours weekly at $8.00 per hours.

Fifty specialists average three to six hours weekly at $10 to $15 per hour.

The seven chiefs of the subspecialty services average three to four hours per week and receive $20 per hour, and the directors of medical and surgical services average three hours per week and receive $25 per hour.

At the above hourly rate, if the general practitioners worked full time—35 hours per week—they would receive approximately $14,000 per year, and the specialists, on the same basis, would receive approximately $20,000 per year. Thus, these physicians are apt to earn much higher *net* incomes on the average than those in individual practice. Fees paid for physician services in the health center in the last year were approximately $235,000.

Payments to Physicians for Services Outside the Health Center

General practitioners are paid $10 a house call for rendering first-aid treatment and/or to determine the need for hospitalization. Any further home care required is not rendered by the health center.

Specialty consultants are paid $25 per patient visit when seen in hospital or private office. Specialists on medical cases in hospital receive $5 per hospital visit, and on surgical cases in hospital are remunerated from health center funds in accordance with our fee schedule, which is somewhat similar to the New York City Blue Shield Schedule of Allowances. Last year the average physician fee per surgical patient amounted to $102 and per medical patient $55.

Total fees for services paid to physicians for medical/surgical services outside the health center during the past year amounted to approximately $100,000. The total amount paid to physicians both in the health center and outside was approximately $335,000 for the year.

Cost of Services

1. *Cost of Medical Services*—The average cost per patient visit to the health center was $6.30.
2. *X-Ray Services*—There were 16,922 x-ray

services, with an average of 1.4 per patient, and the average cost per case was $4.25.

3. *Laboratory*—There were 34,938 laboratory services, with an average of 2.9 per patient, and the average cost per case was $3.42.

4. *Physical Therapy*—There were 14,963 physical therapy and rehabilitation services to 1,007 different patients, with an average of 15 services for each patient, at an average cost per case of $31.33.

5. *Pharmacy*—There were 34,806 prescriptions filled, at cost to the patient, with an average number of prescriptions per patient of 2.9, and an average cost per prescription of $1.35.

Summary of Total Cost of Medical Care

Professional care—medical and surgical		
At the Health Center	$512,532.00	68.2%
Away from the Health Center	116,218.00	15.5%
X-ray	50,651.00	6.7%
Laboratory	40,840.00	5.4%
Physical therapy	31,553.00	4.2%
	$751,794.00	100 %

	Annual cost
Cost per patient	$63.00
Cost per eligible employee	$22.44 (or $1.85 per month)

Summary

As a result of the agreement reached through collective bargaining by the Hotel Association of New York City, Inc., and the New York Hotel Trades Council, AFL-CIO, the hotel workers in 1944 became the beneficiaries of a health and welfare program that provided various forms of insurance, hospitalization through Blue Cross, and certain sickness and accident benefits. After four years' experience with the plan it was further agreed to provide a direct medical service program, financed by the employers and administered by a joint board representing the employers and the hotel trade unions. The plan covers 34,000 active employees of 185 hotels, as well as retired and pensioned union members.

The center of this service program is a health center which provides comprehensive medical care at the ambulatory level. Emergency house calls and hospital care are provided by general practitioners and specialists respectively, who are affiliated with the program. The health center also refers patients to consultants for various specialized office procedures. Hospitalization in hospitals in New York City boroughs is provided for acute medical and surgical conditions and for maternity cases.

The active staff of the health center comprises 17 general physicians and 6 internists, working part-time on regular schedules; an additional 57 physicians serve in specialty clinics for more limited scheduled periods of time. Those physicians who provide house calls, office consultations, and hospital care on a fee-for-service basis bring to a total of 180 the number of physicians formally connected with the program.

The cost of all physician services in the past year was $628,750 (84% of the center's operating cost), and the cost of x-ray, clinical laboratory, and physical therapy procedures was another $123,000 (16%). The cost per patient averaged $63.00, and the cost per eligible employee was $22.44 ($1.86 per month).

The services of the health center itself, are described in some detail, presenting the various phases of the over-all medical care program.

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ROCHESTER (NEW YORK) CHAMBER OF COMMERCE

announces

THE SECOND OCCUPATIONAL HEALTH CONFERENCE OF WESTERN NEW YORK STATE

October 27, 1960

Speakers at the morning session which will be devoted to the subject of rehabilitation, will be Henry Kessler, M.D., Leo Jivoff, M.D. and Sidney Robbins. At the luncheon meeting, Patrick Higgins of the Buffalo Pipe and Foundry Company will describe the advantages he finds in utilizing the community medical facilities in place of a formal medical department. In the afternoon, Robert Sutherland, M.D., of the Ontario Department of Health, will discuss absenteesm, and William Buckley, M.D. will speak on the problem of dermatitis in industry. Each session will be followed by a question and answer period.

Additional information may be obtained from George A. Hardie, M.D., 343 State Street, Rochester 4, N.Y.

How Deep Is Dangerous?

Captain Harry J. Alvis (MC), USN

HOW DEEP is dangerous? This question is not an unusual one these days. The rapid spread of sport diving has placed many aquatic enthusiasts in the way of diving hazards. The well-trained diver knows that diving does not have to be deep to be hazardous. This is an attempt to emphasize the point that it is not the depth so much as the lack of understanding that marks the margin of safety in diving. The reports of diving casualties seen at naval installations in a period of approximately three and a half years were sorted; 20 cases resulting from dives of less than 50 ft. were found.

On theoretical bases, dating back to the work of Haldane in 1909, it should be possible to surface without decompression from a dive of indefinite duration to 33 ft. (pressure of 2 atmospheres absolute). Practical experience proves this is not the entire story. Cases of decompression illness following shallow dives have been reported by Welham and Waite.[1]

For this review, a depth of 50 ft. (pressure of approximately 2½ atmospheres absolute) was chosen because so many sport divers seem to think this is a depth of very little or no hazard. The 20 casualties comprising this review were using a deep-sea diving rig in five cases, a shallow-water face mask in five cases, an open-circuit SCUBA in three cases, and a closed-circuit (oxygen) SCUBA in seven cases. They will be presented according to the equipment used.

Deep-Sea Diving Rig

It would appear that a deep-sea diving rig used at shallow depths should provide the maximum safety. One diver working at depths between 20 and 35 ft. made four dives for a total of about 360 minutes' bottom time (time from leaving the surface to time of leaving the bottom). He developed sharp pains in the shoulders requiring recompression treatment, which gave complete recovery.

Comment—It would appear on theoretical bases that no decompression was required here, but obviously the theory did not hold true. Other possible factors in this case were inaccuracy in depth estimates and the complication of repetitive diving.

A second case was that of a diver working hard at 30 ft. for one hour. To keep himself "heavy" and facilitate maintaining his position at the job, he used only limited ventilation. After an hour, he reported feeling lightheaded, nauseated, and dizzy. When he was brought to the surface, the physical examination added only the observation that the diver was retching without producing any vomitus.

Comment—In all probability, this was a case of carbon dioxide toxicity resulting from restricted ventilation and hard work.

Three episodes were reported following dives in the 41-to-50 ft. range. One case followed a 67-minute dive to 42 ft. Shortly after surfacing, the diver reported abdominal pain and numbness below the umbilicus. This was the second such episode for this diver within a year, each with subjective neurological implications, following what should have been routine dives. Supervisors reported he was an "uneasy" diver. The medical officer disqualified this man from diving.

Comment—Diving, at best, is dangerous work. Anyone who cannot cope with the challenges it presents with mental composure should seek other work. No one will ever know whether this diver really needed recompression treatment or not, despite the fact that he reported he felt free of symptoms afterward. Since this was a poor liability risk, it was proper to disqualify this diver.

The other two cases represent the problem of repetitive dives. One person made two dives to 45 ft., each of 30 minutes' duration. He reported pains in the hip and head. Poor muscular coordination was observed. The other

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The opinions and assertions contained herein are those of the writer and are not to be construed as the official view of the Navy or of those in the Naval service.

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diver made two dives to 50 ft., each of 45 minutes' duration. He complained of pain and mild weakness of the right shoulder and arm. Both of these men were treated by nonmedical diving personnel, with reported relief.

Comment—No comment is justified on medical grounds, since no medically trained personnel saw these patients. It should be pointed out that, in the lore of diving, repetitive dives (more than one a day) have a poor reputation.

Shallow-Water Diving Mask

The shallow-water diving mask is such a simple device that it has led many a curious swimmer into trouble. It lacks the foreboding aspect of the ponderous deep-sea diving rig. The user appears to be a swimmer. The face mask and light hose would appear to be a simple device to use. Most novices overlook the most important fact that anyone breathing a compressed medium underwater is exposed to the hazards of diving.

Two divers reported pain in the left elbow as the only symptom. Both were cured by recompression. One diver had been doing mild work at 50 ft. for 45 minutes. The other had been doing heavy work at 46 ft. for two hours.

Comment—The experienced diving medical officer is not surprised by such seeming variations between individuals. By knowing the diving team with which he works, he learns which divers need more time to decompress and to adjust decompression schedules according to the strenuousness of the work. There simply is no substitute for experience.

A recently trained diver making his first working dive at a new station worked for 13 minutes at 28 ft. He noticed aching in his flanks, dizziness, and difficulty in breathing. His dive was terminated, and he was sent to his bunk to rest. Severe pain on deep inspiration developed in the right chest. A physician experienced in diving was called. He found no objective evidence of anything wrong, but in view of the pain and an uncertain history he tried recompression. This gave relief.

Comment—Did this man hold his breath while surfacing and have a mild air embolism or pulmonary emphysema? Was this hysteria? No one can be certain.

An officer trainee was sawing metal at 25 ft. After 12 minutes' bottom time, he failed to respond to the "How are you?" signal. He was brought to the surface, and it appeared to the tenders that he was unconscious. His air-supply valve was barely open. It was opened wide, and by the time the diver was brought aboard the diving float he was conscious. No further treatment was required.

Comment—This probably was a case of carbon dioxide poisoning resulting from the combination of moderately heavy work and inadequate ventilation. This case illustrates the importance of divers having alert knowledgeable tenders. Generally speaking, other divers make the best tenders because they are better able to imagine what is going on down below. A death by drowning or suffocation was narrowly averted here—thanks to the tender.

In this instance, the diver was working at 10 ft. in water at 36° F. He was being buffeted about by heavy surf and undertow. Eight minutes after he entered the water, the air supply suddenly cut off, presumably because of a kink in the air hose. While attempting to ditch his weight belt, the diver inadvertently knocked his face mask loose. With no air to breathe and being unable to ditch his gear, he gave the emergency signal before he lost consciousness. Although at shallow depth, the diver was some 50 ft. from his tender. The tender got the emergency signal and hauled the diver in. When brought to hand, the diver was cyanotic and not breathing. Artificial respiration was started immediately while others cut the diver's dress from the body. A resuscitator at the scene was put into use. Complete recovery was the result.

Comment—This speaks for itself: Training—Alertness—Preparedness—A live diver.

Closed-Circuit SCUBA

The following seven cases implicate a widely known Italian-made oxygen-supplied self-contained underwater breathing apparatus. None of the divers was reported as being deeper than 30 ft., probably because of an administrative order limiting the use of the equipment to this depth. This figure should not be regarded as scientifically accurate. These cases will be recorded in the timewise order of incidence.

This case resulted from the shortest duration of all and occurred about five minutes after the start of the underwater swim. The patient became dizzy and tried to purge the rebreathing bag. He lost consciousness. His swim buddy inflated his emergency bag, brought him to the surface, and removed the mask. The patient was hyperventilating and unconscious. Small twitching around the right eye and mouth was noted. After several minutes in fresh air, the patient regained consciousness but had a severe headache. (In another exercise by the same group, 5 of 18 swimmers failed to complete the swim. All showed labored breathing, dizziness, nausea, and headache. Two of the five lost consciousness.)

Comment—These men were trying to swim

too fast. CO_2 production exceeded CO_2-absorbing capacity of the canister.

A man was swimming underwater. After 15 minutes, he began to swim faster and shortly thereafter lost consciousnes without warning. When pulled from the water he was confused, dizzy, and nauseated.

Comment—This was caused by CO_2 toxicity and his swimming too fast.

A swimmer had been swimming underwater for about 30 minutes when he noticed a "drawing up of his thighs." His swim buddy observed a generalized clonic convulsive seizure, and brought him to the surface and removed his mask. The patient was unconscious for 15-20 minutes and coughed up blood-tinged froth.

Comment—This was probably an instance of oxygen toxicity, although the possibility of air embolism had to be considered because of bloody sputum.

A swimmer first noted cramps of the feet followed rapidly by doubling up of the legs and drawing up of the thighs, shortly ending in a generalized convulsion. The onset was about 30 minutes after he had commenced an underwater swim. When brought to the surface, he was unconscious and not breathing. Breathing started immediately after artificial respiration was started. Consciousness was regained in about 20 minutes.

Comment—This was an instance of oxygen toxicity.

A man had a generalized clonic convulsion as he surfaced from an underwater swim. He was supported by his buddy until help arrived shortly. The patient was unconscious and threshing about, with bloody froth coming from his nose and mouth. Because of the possibility of air embolism, the patient was taken to a chamber and recompressed.

Comment—This situation confronts the diver's doctor with a dilemma. Does the blood-tinged froth arise from a pulmonary tear? Is air embolism the cause of the unconsciousness? There is no way to tell within sufficient time to prevent serious consequences of cerebral anoxia if a bubble is obstructing circulation. The proper course, as was chosen in this case, is to recompress and observe developments. Subsequent events established that this was a case of oxygen toxicity. The blood came from intraoral lacerations not readily seen and presumably incurred during the convulsion.

This case occurred in one of a pair of buddies who were swimming underwater together. Immediate contact was lost because of poor visibility. When the buddy found this patient, he was on the bottom, 42 ft. deep, trying to put his mouthpiece back in his mouth. The patient was convulsing as his buddy brought him to the surface. Bubbles were observed coming from his mouth during the ascent. At the surface, the patient was comatose and remained unconscious for about 10 minutes.

Comment—This was another instance of oxygen toxicity, all right, but where was the buddy line? SCUBA divers should *always* dive in pairs and should be fastened together by a buddy line when diving in waters and conditions of limited visibility. A drowning was narrowly averted in this instance.

This man had been swimming underwater for 30 minutes uneventfully. Then he experienced three bouts of respiratory difficulty similar to diaphragmatic spasm, each relieved by rest and purging. (As used here purging refers to emptying the rebreathing bag and refilling with a fresh supply from the oxygen bottle.) A fourth episode of respiratory difficulty occurred after 57 minutes of swimming and could not be relieved by rest and purging. During ascent to the surface, the patient became unconscious and experienced generalized convulsions. The tonic-clonic seizure persisted for six minutes after the patient was placed in a boat. There was post-ictal confusion and hyperexcitability for 30 minutes. Recovery was complete.

Comment—This was probably oxygen toxicity. Diaphragmatic spasms are experienced by skin divers when they are approaching the limits of a breathhold. This can occur at depths when the alveolar oxygen partial pressure is still well above the level normally present. Presumably this represents a damming back of metabolic carbon dioxide. In this case, the spasms probably represent an unusual site for the muscular twitching, which is a fairly common prodromal sign of oxygen convulsion.

Open Circuit SCUBA

The first case of this grouping ended tragically in a pond of water not more than 20 ft. deep, according to the report. Two SCUBA divers entered the muddy waters of a pond to rearrange some heavy equipment located in the water. The divers became separated; one surfaced and looked around for the bubbles from his buddy. Seeing none, he knew something was amiss, dove again immediately, and searched for the buddy. The buddy was found on the bottom with the face mask off (both were using full face mask open-circuit air-supplied SCUBA). The diver brought his drowning buddy to the surface, but in doing so he dislodged his own mask, so that it flooded and he could not breathe. He could not manage his own difficulty and support his buddy with

one arm, and so the buddy slipped to the bottom of the pond. The diver then took a line down, found his buddy a second time, and secured the line to him, whereupon those on shore were able to assist in bringing the drowned diver to the shore, unfortunately too late.

Comment—The usual and proper practice of using a buddy line in reduced visibility was not adhered to in this instance because of the presence of numerous rigging lines already in the pond. Presumably, the diver became entangled in some of the rigging and had dislodged his face mask. Then he was unable to locate it, clear it, and replace it before he suffocated and drowned. The rescue was complicated by the difficulties in twice locating the diver in trouble. It also appeared that the drowned diver might have tried to ditch his equipment but could not manage to free himself because of an inadequate length of strap for a handhold.

This patient was swimming at various depths down to 48 ft. for an hour. After surfacing, he had some soreness over the anterior chest wall, which was regarded as myositis and which disappeared in about six hours. Twelve hours after the dive, the patient noted mild aching spread over the lateral aspect of the right arm with a weak grasp on the right. A trial of recompression relieved the symptoms and cured the patient.

Comment—One would not expect a case of decompression sickness (bends) from a dive at this depth of this duration. Perhaps this simply illustrates the wide variation in the uptake and release of inert gas.

An 18-year-old self-trained SCUBA diver was doing some light work at 40 ft. for about two and a half hours when he became fouled. In freeing himself, he broke his supply hose; so he exhaled forcibly and swam to the surface. He immediately felt dizzy and weak and noted some blood in his mask. A few minutes later he noted aches in his chest, knees, and shoulders which were partially relieved by cold applications. The next morning he still ached and felt lightheaded; so he sought medical aid. He was flown to a recompression chamber. When seen by a diver's doctor, the only positive evidence were a mottling of the skin over the shoulders and the patient's report of scattered aches and substernal pain. Although this was a vague array of symptoms at best, a decision was made to try recompression. This brought complete relief at a simulated depth of 50 ft.

Comment—Here again is a dive that would not ordinarily be expected to cause decompression sickness. Two and a half hours spent at 40 ft. in the water off Long Island (New York) in early September is not exactly a warm bath, but one would expect the effects of chilling to clear up overnight. One might assume the relief obtained from cold applications was simply cryogenic analgesia. It is not adequate treatment for decompression illiness.

Discussion

This series of 20 cases should convince any reasonable person that depth is only one of the many considerations in diving. No one kind of equipment is without hazard. On the contrary, each kind of equipment has its own peculiar array of hazards in addition to the general hazards of diving. Among the general hazards of diving, this series illustrates inadequate decompression, carbon dioxide toxicity, oxygen toxicity, air embolism, and drowning. There was no clear case of nitrogen narcosis (because of the shallow depth limiting the nitrogen partial pressure) nor of "diver's squeeze" or "blow-up."

In general, decompression illness from dives to this depth would not be expected to occur. Elements that modify this generalization and may be the explanation of some of these cases are inaccuracies in estimates of depth and duration of the dive, the severity of the work, the temperature of the water, the equanimity of the diver, the age of the diver, and repetitive diving. Any and all of these variables may influence the uptake and elimination of inert gas—the culprit in decompression illness. The decision whether or not to recompress is often solved by the lack of faciilties. When facilities are available and knowledgeable medical supervision is at hand, recompression may be tried without any great hazard to the patient. The lapse of time between onset of symptoms and recompression greatly influences the prospects of cure. Rarely is recompression helpful after a lapse of 48 hours.

Carbon dioxide toxicity was illustrated by several of the cases. In the deep-sea suit or the shallow-water face-mask rig supplied with air, this becomes a question of ventilation through the helmet or mask sufficient to sweep out the exhaled carbon dioxide. In the closed-circuit oxygen-supplied SCUBA the condition can arise on several bases. Any closed-circuit breathing equipment requires some provision for removal of exhaled carbon dioxide. This removal can be inadequate because of improper design of the canister so that there is insufficient absorbent surface exposed, debilitation of the absorbent chemical, or accumulation of moisture by condensation or leakage, or simply because the diver swims too fast or works too hard so that carbon dioxide is produced in

excess of the absorbing capacity of the canister.

The operation of closed-circuit SCUBA deserves further explanation. This equipment is essentially a closed circuit of a rebreather bag and the diver's lungs with a carbon dioxide removal canister in the flow between the bag and the lungs. Some of these have a valve arrangement to keep the breathing medium flowing in a circle. Others accept to-and-fro (pendulum) breathing. An oxygen bottle is connected somewhere into the system, from which the diver can replenish the oxygen as it is consumed. When the equipment is donned, the bag is inflated with enough oxygen to provide a countervolume to the lung volume. As the diver proceeds breathing oxygen, the normal sea-level nitrogen content of his body flows into the rebreathing bag because it, being filled with oxygen at the start, presents a reduced partial pressure of nitrogen at the pulmonary membrane. As the diver proceeds, the oxygen in the bag is used up and to some extent is replaced with nitrogen leaving his body.

Unless forewarned of this hazard and properly trained, the diver can pass, unaware of his dilemma, into anoxic unconsciousness. The remedy is to periodically deflate (purge) the bag as completely as possible and refill it with oxygen. The possibility of gradual anoxia as a result of an oversight somewhere in this procedure is a frightening prospect; and when to this is added a possible failure to provide sufficient carbon dioxide absorption capacity, the potential hazard becomes overwhelming. A further consideration is that oxygen can be a convulsant, a fact not widely known even in the medical profession. This effect depends on the partial pressure and length of exposure. Depth increases the partial pressure. Using oxygen-supplied equipment increases the partial pressure. It appears probable that elevated tissue carbon dioxide increases the liability. An oxygen convulsion is just like a grand mal seizure in appearance. Obviously this is not the sort of thing to court underwater. It is for these reasons that knowledgeable diver-doctors so strongly advise against any improvised, do-it-yourself-built SCUBA of the closed-circuit oxygen-supplied variety. This piece of equipment has no place in sport diving.

The diver's best friend is his tender. A good tender must be able to imagine what is going on below. Two cases in this series demonstrate the lifesaving dependence on the tender's alertness and knowledge and the related preparations and training. In SCUBA diving, the buddy is the tender. Each diver of a pair tends (observes) his buddy. When visibility is limited, there is no excuse for not using a buddy line to secure the divers to each other. Only those who have felt the heart-cramping grief of fruitless searching in muddy water or have felt a friend slip from their grasp can fully appreciate this advice. A six-ft. length of sash cord is so cheap, and that is all it takes. Rules for the SCUBA diver are: DON'T DIVE ALONE, and DON'T EVER FAIL TO USE A BUDDY-LINE WHEN IT IS INDICATED.

Any diver using lightweight equipment that can become fouled or disarranged must understand how to free himself from his equipment and how to make a safe ascent. As long as air is available from the supply—hose or tank—it should not be discarded. The diver should stay with it even though it is entangled, until he is certain he is free. If he must abandon his equipment, he *must* exhale as he rises to the surface. The wearing of flotation equipment deserves encouragement. It can bring the diver to the surface and help keep him afloat at the surface.

No one laughs at fire escapes on buildings. No one feels content in a building from which he could not escape in case of fire. For exactly the same reasons, no SCUBA diver should wear equipment from which he cannot escape. Practice ditching of equipment should be part of every training program. Other cases of drowning than the one reported here have occurred where a SCUBA diver drowned because he could not get loose from his equipment. There are ways to thread the straps of a harness so they can be jerked loose quickly, but the end must be long enough to afford a good hold. There are quick-release—type of catches for SCUBA harness, but an emergency situation may be too late for a practice session. The SCUBA diver must know how to ditch and ascend safely.

Any time a case of decompression illness or air embolism is flown to a recompression chamber, the flight should be made at the lowest safe altitude. As flight altitude increases, total pressure decreases, and any bubble that has formed expands. In the event no recompression facilities are available, a theoretically useful therapeutic measure is to administer oxygen. This must be continued for several hours to be meaningful. Oxygen does not relieve the symptoms resulting from obstruction of blood flow by a bubble so dramatically as compressing the bubble to smaller size by recompression, but it does promote the elimination of inert gas from the body. Since the gas tension in a bubble is in an equilibrium with the gas tension of the surrounding tissue, the bubble will shrink as the inert gas is washed out of the tissues.

Reference

1. Welham, W., and Waite, C. L.: Decompression Sickness: Report of Two Unusual Cases, *Armed Forces Med. J.* 2:1201-1204, 1951.

Suggested Reading

Diving Practices

1. Submarine Medicine Practice (NAVMED P-5054), Washington, D. C., U. S. Government Printing Office, $2.00.
2. Part III, U. S. Navy Diving Manual (195), Washington, D.C., U. S. Government Printing Office, $2.00.
3. Science of Skin and Scuba Diving, C.N.C.A., New York, Association Press, 1957, $3.98.

Medical Aspects

4. Lanphier, E. H.: Diving Medicine, *New England J. Med.* 256-120-131 (Jan. 17) 1957.
5. Alvis, H. J.: Hazards of Diving with Self-Contained Underwater Breathing Apparatus, *Indust. Med. & Surg.* 27:392 (Aug.) 1958.
6. Duffner, G. J.: Medical Problems Involved in Underwater Compression and Decompression. *Ciba Symposia* 10:4 (July-Aug.) 1958.

INDUSTRIAL HYGIENE FOUNDATION

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MANAGEMENT CONFERENCE

Group Insurance—A Mounting Production Cost
Let's Stop the Misuse of Medical Money—E. T. Klassen
Group Insurance and Health Care Costs—R. R. Shinn
Sickness and Absenteeism Can Be Controlled—J. F. McCahan, M.D.
Effects of Food Additives Legislation on Food Technology: Industry's Viewpoint—K. E. Mulford; Legal Aspects—L. A. Coleman; Public Welfare—D. B. Hand
Standards for Air Quality—V. G. MacKenzie

JOINT MEDICAL-LEGAL CONFERENCE

Emphysema, Pneumoconioses and Compensation: Clinician—O. A. Sander, M.D.; Pathologist—A. J. Vorwald, M. D.; Physiologist—G. W. Wright, M.D.; Attorney—W. C. Hartman

MEDICAL CONFERENCE

Methods of Measuring Work Capabilities of the Individual
Information That Is Needed: Responsibilities of Medical Scientists and of Industrial Physicians—H. S. Belding, Ph.D.
Physiological Approaches to Evaluation Capabilities—W. H. M. Morris, Ph.D.
Psychological Approaches to Evaluational Capabilities—L. W. Gregg, Ph.D.
Usefulness of Procedures of Health Examinations—W. D. David, M.D.

LEGAL CONFERENCE

Legislative Developments in Workmen's Compensation for the Year 1960—Andrew Kalmykow
Some Legal Aspects of Compensation in Heart Disease Cases—H. F. McNiece
Problems Presented to Directors of Departments of Industrial Relations in the Handling of Workmen's Compensation Claims—O. T. Clayton
Workmen's Compensation in Foreign Countries—Ivan Sabourin

ENGINEERING CONFERENCE

Acceptable Equipment and Procedures for Evaluating Air Pollution Problems—R. O. McCaldin, Ph.D.
Environmental Performance Standards: Their Place in Planning and Zoning Regulations—W. T. Ingram
Benefits of Central Gas Cleaning Facilities—C. A. Bishop, Ph.D., and G. A. Howell
How Can We Achieve Better Operation of Dust Control Systems—K. J. Caplan
Recent Welding Practices—Naval Facilities—C. P. Bergtholdt
Legal Considerations Involved in Developing Precautionary Labels—J. D. Kittelton
A Progress Report on In-Plant Hazard Identification Systems—J. J. Duggan
Recent Developments in Industrial Hygiene: Plasma Jet—H. W. Speicher; Fuel Cell—R. H. Blackmer; New Epoxy Resins and Cutting Oils—N. G. White, Ph.D.
Some Recent Applications of Glass Cloth Collectors for High Temperature Gases—R. J. Wright and R. T. Pring

CHEMICAL-TOXICOLOGICAL CONFERENCE

Pulmonary Edema and the Permeability of the Alveolar Membranes—J. A. Clements, M.D.
Toxicologic Mechanisms at the Cellular Level—C. J. Kensler, Ph.D.
Chemical Antidotes for Radiation Injury—John Doull, M.D.
Fuels and Their Combustion Products: Diesel Fuels—J. F. Morgan; Missile Fuels—E. B. Rifkin, Ph.D.; Rocket Fuels—H. M. Bowman

The Treatment of Industrial Hand Injuries

I. GENERAL PRINCIPLES OF WOUND TREATMENT AND INJURIES TO ENVELOPING TISSUES

Burton C. Kilbourne, M.D., and Eudell G. Paul, M.D., Chicago, Ill.

DISABLING hand and finger injuries occur yearly at the rate of 450,000 in industry, and constitute 25% of all work injuries. From the same statistical source can be projected 825,000 hand and finger injuries of farm and nonwork origin annually. Even though these figures represent an improvement over the rates of 30 years ago, their magnitude commands a continuing interest in prevention and treatment of hand injuries.

To maintain high standards of treatment, basic principles of early, adequate care must be emphasized constantly. Although new techniques are to be sought, they must be critically evaluated. The entire medical team should study causes of infections and failures. It is particularly desirable that we recognize the functional and economic loss in the mishandled or neglected case.

Care at the Plant

From the inception of injury until the time of definitive surgical treatment, an employee is seen and evaluated by several of the industrial medical team. The fellow workman or foreman should know that his first aid is to provide rapid covering of the wound with clean towel or dressing. He can offer encouragement and accompany the more seriously injured to the plant dispensary or doctor's office. The nurse or physician will then evaluate the situation for treatment. They must know their limitations in equipment and facilities, and not underestimate the requirements of an injury which may be best treated at the hospital. A history is obtained, and visual and x-ray examinations are performed. Should the plant or office facilities be adequate, treatment is undertaken immediately. For the more serious injuries, such as amputation, open fracture, extensive laceration, and severed tendon or nerve, the use of a hospital operating room is indicated. In such instances, the procedures at the plant should be limited to giving medication for relief of pain, control of hemorrhage by pressure dressing, and application of a splint. By prompt communication with the surgeon and hospital, the elapsed time from inception of injury to definitive operative repair will be minimized. Too frequently there is delay between plant care, hospital admission, and definitive surgery. The outcome of an otherwise good repair may be jeopardized by this delay. We wish to reemphasize the importance of an effective nursing organization.

Mechanism of Injury

The mechanism of injury is important. Various mechanisms imply degrees of severity as well as specificity of tissue injury. The guillotine action of cutters and presses leaves little to repair but a digital or hand stump. The variable penetration of a power saw determines the need for skin, tendon or nerve, and fracture repair or amputation. Puncture by a drill or a sharp punching tool leaves only a small skin wound, but a digital nerve or flexor tendon may have been severed. Flying metal chips and wood and glass splinters enter through small wounds to become imbedded at any depth in the tissues. Roller and wringer injuries are known to show only slight early evidence of injury, to be followed later by skin necrosis. This is a result of intense venous congestion followed by thrombosis or is due to subcutaneous disruption of vascular channels and hematoma. A hot roller or mangle compounds this effect by the superimposed burn. The abrasive action of a grinder produces a burn and a laceration to variable depths. The necrotizing effect of grease-gun injection into hand tissues must be appreciated in order that early

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radical drainage can be provided. The mechanisms of closed injuries will suggest which structures may be injured. The patterns of injury in a given plant or industry should be recognized for their specific potentialities. Obviously many of these injuries involve multiple structures and require skill in diagnosis and treatment of proportionate magnitude.

In our discussion we will consider the principles of wound treatment applicable to all injuries, and follow with specific handling of injuries to the skin and covering tissues, the tendons and nerves, bones and joints, and amputations. It is not practical to cover every possible combination of multiple tissue injury, but we wish to stress principles for each that will be applicable alone or in combined injuries.

General Principles of Wound Treatment

In most instances, it is not only possible but desirable to convert the contaminated industrial hand wound to a clean wound and safely accomplish primary wound closure. This presumes adherence to a rigid technique.

We must avoid further contamination of the wound. Caps, masks, and gloves are always worn when treating any hand wound about whose severity there is some question. With sterile dressings covering the wound, peripheral examination is done. Sensation is appraised, tendons are tested against resistance, and the integrity of bones and joints is determined. Appropriate drugs are ordered.

Then, with the treatment team wearing caps, gowns, masks, and gloves and in the operating room, the wound is carefully cleansed. The extent of the injury determines the need for local or general anesthesia. With the open wound covered with a light sterile dressing, the surrounding skin is cleansed with soap and water. Initial removal of grease, oil, tar, etc., with an appropriate solvent may be necessary. Finger nails are trimmed and freed of underlying dirt. The wound itself is not cleansed with soap and water but is irrigated with copious amounts of sterile isotonic saline. The application of skin disinfectants is an individual preference.

After regloving and gowning, the patient's hand and arm are draped. To obtain a bloodless field, a pneumatic tourniquet or blood pressure cuff inflated to 280 mm Hg is used. It is applied evenly to the arm, which has been covered by a few turns of soft sheet wadding. As the debridement proceeds, it may be advantageous to release the pressure from time to time to check the vacularity of flaps. When the cuff is being reinflated, the extremity must be adequately drained of blood by elevation for 60-120 seconds or by the use of a Martin bandage. Tourniquet paralysis is very real. An arbitrary time limit of two hours of compression has been set with an allowance of 15 minutes to flush out the extremity. We feel that improper use of the pneumatic cuff and failure to control the cuff pressure (either too little or too great) are responsible for the trapping of venous blood and subsequent thrombosis. In well over 1,000 cases we have had but one such catastrophe. (Fortunately, full recovery followed with such supportive measures as splinting and physical therapy.)

Actual debridement is carried out with fine plastic surgery instruments comparable in size to the well known mosquito hemostat. Sponges and dressings used during the procedure should be moistened with sterile saline. Tissue dryness adds to the traumatic insult.

We must eliminate contaminants. Debridement is the deliberate and meticulous excision of all devitalized tissue and of all tissue destined to succumb. En bloc excision is not implied; viable tissue is not sacrificed. One would best develop a routine of excision. Only the narrowest skin margin or none at all is taken in cleanly incised wounds, but severe crushing injuries may necessitate the removal of large segments. Needless to say, nerve and tendon are conservatively treated unless irreversibly crushed. Tendons may be "shaved" of contaminants. Dark, dusky, and noncontractile muscle is removed. Debridement goes hand in hand with exploration of the deeper aspects of the wound. One must not hesitate to extend the wound by appropriate incision in order that adequate exploration be accomplished.

Injuries to Enveloping Tissues

Although injuries which involve the hand are likely to disrupt multiple structures, there are some in which trauma to the skin will be predominant. The following common industrial injuries will be discussed in order: abrasions, punctures, lacerations (incised and avulsed), crushing, and burns.

Abrasions—In this injury, the velocity of the causal agent will determine the depth, contamination, and extent of the wound. A simple abrasion requires nothing more than gentle cleansing followed by a light dressing with or without a bland innocuous ointment. When the skin is abraided by a stationary object, thorough scrubbing and scraping should be done but not to the production of a full-thickness defect, as it is well known that these lesions heal with mere protection and rest. It is important to scrape or sand the wound

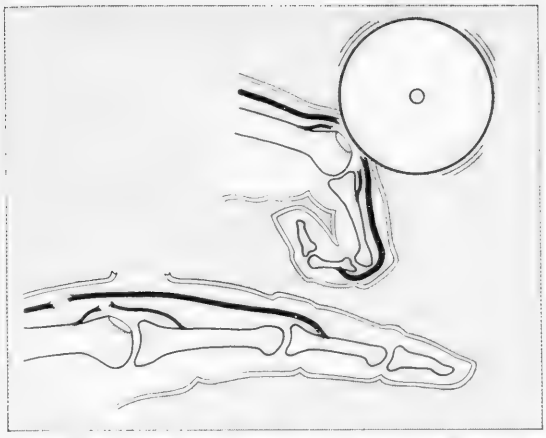

Fig. 1—Depicts the shifting of structures in a grinding-wheel injury when the position of the finger changes. Unless the surgeon is aware of this mechanism, the true extent of the injury will be missed.

in which is imbedded coal dust or other carbonaceous material capable of tattooing the skin. Those abrasions produced by power equipment at higher speeds may often present a full thickness defect (an abrasion plus burning [heat] destroys tissues.) When the skin is not penetrated, the appearance is often as deceiving as a burn and a full-thickness slough can ensue. If this is suspected in a wound of more than small proportions, the wound should be excised and the defect closed by approximation of adjacent skin margins or by application of a skin graft. The decision to excise an injured area will rest in its location, its size, and the estimated length of time to heal. If it can be expected to heal in three weeks, excision and graft are probably not indicated. Our experience has shown that an area over 3/8 inch in diameter will normally not heal in three weeks and will be delayed well beyond this period. Less temporary disability will ensue if excision and plastic repair are carried out.

In an open wound produced by a grinding wheel, complete excision is necessary, for the "grind" burns and imbeds minute emery particles in all tissues it touches. Any grind over the more vulnerable dorsal aspect of the proximal interphalangeal and metacarpal-phalangeal joints can be a most treacherous one. Here the shifting of tissues with changes in position of the fingers conceals underlying involvement. Because of this mechanism it is easy to fail to recognize the involvement of an extensor tendon or a joint surface (Fig 1). The technique of wide excision and closure of these wounds will be discussed under avulsions.

Puncture Wounds—The decision to treat a puncture wound surgically is influenced by its appearance, extent of the wound, severence of nerve or tendon, joint penetration, and the possibility of imbedded foreign material. The smaller minor punctures may be treated by adequate skin cleansing, fluffy compression dressing, and splinting for rest and protection. A tetanus toxoid booster or, after skin testing, tetanus antitoxin is given. Those puncture wounds which are slightly larger, more than 2-3 mm in diameter, should be treated in a similar manner with the addition of cruciate incisions over the puncture followed by excision of the four skin points so produced. Exploration of large punctures is often necessary, especially if clothing (glove) was penetrated.

A very common industrial hand injury is penetration by a metallic foreign body. Most of such foreign bodies in the hand should be removed, especially if more than 2 mm in diameter and if lodged within the palmar compartment of the hand and fingers. When these are lodged close to the skin, tendons, or nerves, they produce pain and irritation on grasping.

Obviously, those wounds which proceed to frank infection must be treated by an appropriate antibiotic, warm wet dressings, adequate incision and drainage, and further splinting.

Lacerations—(1) Incised: Unless underlying structures are severed, incised lacerations are cleansed with soap and water, irrigated with saline, and sutured with fine interrupted sutures. We prefer 5-0 nylon. All finger and hand lacerations heal more kindly with even compression dressings and splinting.

(2) Avulsed: This type of laceration implies greater contamination, avascularity of flaps, and even some loss of skin, fat, and fascia.

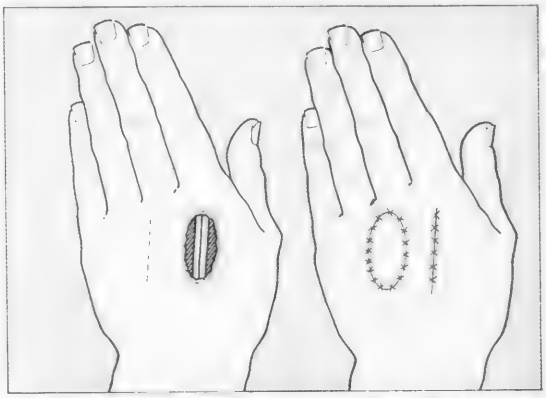

Fig. 2—The figure on the right depicts the appearance of the exposed extensor tendons of the index finger. After debridement, the wound may be closed by means of a relaxing incision and shifting of a full-thickness layer of skin and subcutaneous tissue. The surgical defect is closed with a split-skin graft. A unique rotation of larger local flaps is described by Flatt[2] for smaller defects exposing flexor and extensor tendons in the hand.

The skin edges are likely to be uneven and ragged. After preliminary peripheral examination, skin cleansing, wound irrigation, and assessment of deep injury, one must decide between simple or plastic closure. When there is a viable avulsed flap the wound is prepared for closure by excision of 1 mm of bruised or jagged skin edge and any devitalized underlying fascia or fat.

When a skin defect exists as a result of the avulsion, it may be closed by a primary split skin graft or by immediate transposition of tissues. Application of a skin graft requires a suitable base of fat and fascia, as a good "take" will not be achieved over exposed tendon, bone, or joint. These vulnerable structures will become necrotic and slough unless covered by a full thickness flap of skin and subcutaneous fat.

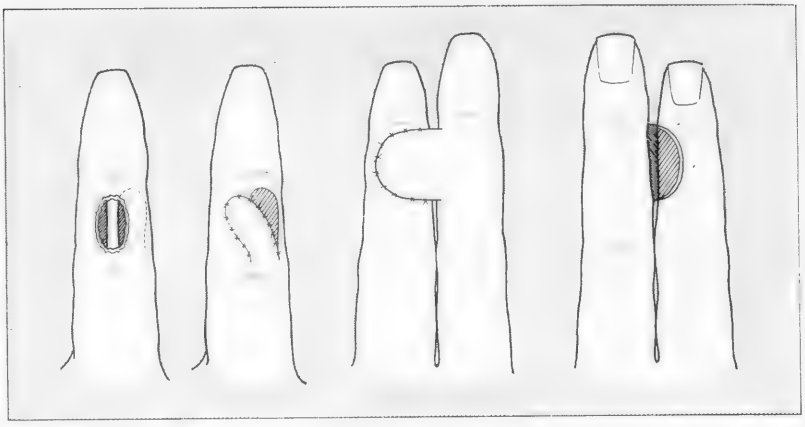

Fig. 3—*A* depicts a small rotation flap used to cover an exposed flexor tendon. The surgeon is cautioned not to trim the buckled corner of the flap; in so doing, its blood supply will be jeopardized. *B* represents volar and dorsal views of a common cross-finger flap. Note that the flap is not raised from the volar skin. Although the fingers are shown in extension, they should be dressed and splinted in a more natural position of function.

More extensive avulsions of dorsal or palmar skin may require removal, defatting of the flap, and resuture (replacement). Wide exposure of bones and tendons will necessitate immediate use of larger distant flaps.

The completely degloved hand poses a perplexing problem in treatment. Three courses of action may be followed. Flap coverage is technically difficult, and is to be done only when some degree of pinch or grasp mechanism remains. One is not justified in subjecting the patient to lengthy initial and subsequent procedures unless he can expect survival and function of at least part of a thumb and one finger. In addition, such hands are often completely lost, owing to marginal vascularity with late thrombosis and infection. Although not a definitive procedure, immediate coverage with free split grafts will often preserve a hand for subsequent reconstruction. A third consideration is initial partial or complete amputation. This, of course, is governed by the degree of underlying vascular and osseous insult. As previously stated, amputation may be indicated if there is no expectation of remaining pinch function.

Various procedures are used to shift skin from one location to another. Elaborately fashioned flaps from a distance are often more detrimental than beneficial to the denuded fingers. Similarly, the elderly and arthritic hand does not do well in any type of flap closure. Rotation flaps from adjacent tissues or cross-finger flaps[1] lend excellent coverage without restricting the hand and extremity unnecessarily. For smaller defects, a rotation or a sliding flap can readily be accomplished. The latter is merely a relaxation incision which allows a full thickness of skin and subcutaneous tissue to be shifted.

In the construction of any flap of tissue, one must exercise gentle meticulous handling of the skin under controlled ischemia. Digital bundles, of course, must be preserved. In selection of a donor site one should avoid skin of the distal phalanx and also skin volar to the midlateral line of the finger; the flap should not cross flexion creases. Some sort of skin marking (methylene blue) will help in planning an accurate flap, as will mapping the defect with paper (Patapar). Flaps should be larger than the defect. When the length is greater than the width, its base should be proximal. The resulting defect produced by elevation of the flap can be closed by the application of a small free split graft taken from the same forearm.

Prior to closure of the defects with 5-0 nylon, the tourniquet should be released and adequate hemostasis secured by ligating vessels with #100 cotton or 5-0 gut. Fluffy compression dressings are then applied and held in place with Elastoplast, and the hand and forearm are immobilized on aluminum splints or in plaster.

Partial change of dressings is done in 48-72 hours, sutures are removed in approximately 14 days, and the flap is sectioned in 14-21 days.

Crushing Injuries—Crushing injuries imply

those which are just short of amputation. Indeed, many fingers so injured will have to be sacrificed. Careful debridement is the key to success in the treatment of these mangling injuries. Tendon repair as an initial procedure is often impossible, but an attempt to anastomose the digital nerves is advisable. If the finger is to be salvaged, skin coverage is mandatory. This can be done with free grafts and transposition of skin flaps. The scarring which occurs in crushed fingers may cause ankylosis in extremely unfavorable position. The treating surgeon can here exercise mature judgment by deciding between amputation or surgical arthrodesis. A more acceptable finger, both cosmetically and functionally, will result.

The arthrodesis often may be accomplished by mere splinting of the finger with intramedullary Kirschner wires (removable). The requirements and technique of this procedure include excision of joint cartilage, proper beveling of joint surfaces, use of bone pegs for strut grafts, and internal splinting in the desired position of function by means of removable intramedullary Kirschner wires. Obviously, external splinting is also necessary for four to eight weeks until fusion can be expected. The intramedullary wires then can be extracted easily without anesthesia.

Burns—Although usually only a part of the extensive injury, burns of the hand are common in industrial, military, and civilian practices. Severe crippling is seen frequently even in fingers and hands that have been properly treated. This, of course, is due to the compactness of structures in a structure with highly specialized kinetic properties. Early skin coverage may obviate an otherwise inevitable deformity and restore useful function.

The more urgent steps in the care of the patient having been initiated, attention is directed toward management of the local wound. Packs of sterile dressings should be stored and readily available in any industrial medical facility. Fine mesh gauze (not roller gauze), nylon mesh, or Furacin-impregnated gauze can easily be kept in sterile containers and can be used also for other types of wounds. Fluff dressings, the equivalent of two to four 5-yard Kerlix rolls, are usually adequate for dressing one entire hand. The rolls may be packaged as rolls or presectioned into 1-foot lengths and "fluffed out" for immediate use. Tensor bandages and splints (peach board, aluminum, or plaster) complete the list of necessary materials.

Strict aseptic technique must be employed in the care of all burns, large or small. Cap, mask, and sterile gloves are to be worn by the treating physician, and at least masks by those attendants in close proximity to the patient. These precautions being observed, the hand is cleansed gently with bland soap and water or with isotonic saline only. Loose grossly contaminated skin may be sharply debrided with scissors; blebs may be evacuated and trimmed. It should be emphasized that the skin is not to be vigorously scrubbed. A single layer of previously selected material is applied directly to the wound. We do not favor gauze heavily impregnated with petrolatum or other materials, for such dressings trap the secretions and do not allow the wound to "breathe." Fluff dressings can be applied evenly to the contour of the hand and provide resiliency rather than tightness. The entire hand and forearm are immobilized on a splint in a position of function. The severely burned hand assumes a grotesque appearance easily enough when not protected by proper splinting. Such a dressing is not disturbed for five to seven days unless one suspects infection or impaired circulation to the fingers. A finger with a circumferential burn may require surgical decompression. It should be emphasized that a burn of an individual finger also deserves splinting of the involved part.

At the first dressing one may observe partial healing of the more superficial burn and separation of the more deeply burned skin. As the slough begins to demarcate and separate, a change to daily wet saline dressings with careful debridement will often prepare the burned surface for grafting by the end of 14 days. A bed of flat young clean granulations is necessary for acceptance of a graft.

There are advocates of early excision of full thickness burned tissue. Ideally, excision is done between 48 and 72 hours, as earlier excision is followed by too much subsequent edema. Removal of skin must be carefully carried out; the proper plane is one just beneath the thrombosed vessels. Since perivascular lymphatics are important, as many open vessels as possible should be saved. One should leave the thin filmy areolar tissue immediately over the tendons. One may graft immediately or delay the procedure for 48-72 hours. The use of a large drum (Padgett) is advisable, since piecing together is less often necessary with the large strip of skin obtained.

Thin split-thickness grafts should be placed over the dorsum of the hand with their margins following a zigzag pattern to help minimize contractures. Skin over the dorsum of the fingers should be brought to the midlateral lines. The interdigital webs can be covered by a

tongue of skin cut into the main graft, carried over the web, and sutured to the palmar surface of the hand.

Fortunately, palmar surface burns are not so common as those of extensor surfaces owing to the individual's protective instincts in shielding himself during fires and explosions.

Having secured the graft with fine nonabsorbable sutures, fine mesh gauze, nylon, or Furacin gauze is placed over the grafted skin. Should there be risk of motion or difficulty in securing the graft to the contour of the hand in certain locations, bolster sutures may be used to tie the dressings in place. The same fluffy compression dressing is then applied to the hand as is used in the initial burn dressing. An elastic bandage can be used for the desired degree of compression. We splint or apply casts to all hands with the wrist and fingers in a position of function. Unless one suspects infection, initial inspection of the graft is not done for three to five days. If hematomata are present, they are evacuated at that time, but one should avoid forceful irrigation and flushing of the undersurface of the graft, lest its entire adherence be disrupted. Should the wound look "soupy" (infected, with free or trapped exudate), one must change to wet dressings (saline). Sterile rubber Dakin tubes are placed within the dressings for intermittent instillation of isotonic saline. Such a wound must then be dressed daily.

The noninfected graft is redressed with compression and splinting which are continued for another three to five days. Sutures may be removed in 12-14 days. We apply a lighter protective dressing after the second week, but usually do not begin soaking and active exercise until the beginning of the third week. Since both trauma and immobilization produce so much fibrosis and stiffness, intelligent and diligent use of physical therapy is of inestimable value in later treatment. With a little instruction, most patients can perform the prescribed exercise at home. Some will require the direct supervision of a physical therapist.

Patients who are sent to a physical therapy unit must be advised to continue the exercises at home; 15 to 30 minutes three times a week is not sufficient. A common deterrent to the continuation of exercises is the pain and swelling which follow attempts to restore finger-joint motion. An informed patient will not allow this to become a delaying obstacle. Early return to work is to be emphasized. Active use in performing purposeful tasks is more important than time itself; this, of course, is true for all types of hand injuries. One must always remember that treatment does not cease with the removal of sutures and dressings or with the patient's return to work.

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Correction

An error appears in the text of the second paragraph of the article "DTPA—Effectiveness in Removing Internally Deposited Plutonium from Humans," by W. D. Norwood, M.D., in the August, 1960, issue of this journal, page 371. DTPA is referred to as trisodium diethylene*tetra*minepentaacetic acid. DTPA is a *tri*amine compound as shown in the molecular structure which appears two pages later in the same article.

The Toxicology of Some Aliphatic Alcohols—Part II

E. V. Henson, M.D., South Charleston, W. Va.

IN PART I of this review the summary of action and general chemistry of the alcohols were considered. The industrial and clinical features of intoxication by some of the common alcohols will be described in some detail in this part.

Methanol

Methanol (methyl alcohol, wood alcohol, carbinol, CH_3OH) is a water-white mobile liquid with a sharp, ethereal odor. It is completely soluble in water, has a molecular weight of 32.04, and boils at 64.5°C. It has extensive applications as an industrial and commercial solvent, anti-freeze, and chemical intermediate.

For decades methanol has been recognized as a poisonous material, although the reason for the observed ill effects was not known definitely. An impurity such as allyl alcohol which is present in methyl alcohol produced by the destructive distillation of wood had been thought to be the offending agent. When pure synthetic methanol became available, the clinical features of intoxication following its consumption remained unchanged, and it became apparent that the previously observed disastrous effects were related to the alcohol itself.

Although methanol poisoning occurs primarily from the ingestion of adulterated alcoholic beverages, symptoms can occur from absorption through the lungs and skin. As recently as 1955 headaches occurred among employees using methanol in duplicating machines in small, poorly-ventilated rooms. Exposures were to concentrations in the order of 300 ppm[1] and 800 ppm.[2] McCord,[3] using several species of animals, was able to demonstrate that with prolonged contact fatal amounts of methanol could be absorbed through the skin. Using the data obtained with monkeys, he estimated that one ounce of methanol absorbed through the skin could constitute a threat to man.

Impairment of vision and death from the cutaneous and pulmonary absorption of methanol have been reported in the older literature.[3-8] Unfortunately, the exposures were not quantified. Reports that the alcohol had been rubbed on the skin for a month or more as a liniment for rheumatism[6] and that prolonged exposure had occurred within a poorly ventilated vat and had been followed by symptoms of inebriation[5] would indicate that large doses were absorbed. Other cases were poorly documented and cannot be accepted as unquestionable, authentic intoxications. The 54 cases compiled by Wood and Buller[4] and the statement by Tyson and Schoenberg[7] that by 1912 about 100 cases of amblyopia and death from inhalation of methanol had been recorded in the literature would certainly indicate that the oral route of administration is not the only way by which harmful quantities can enter the body. A case of chronic poisoning from methanol vapor with marked diminution of vision and enlargement of the liver was reported by Humperdinck.[9] The employee was engaged in centrifuging material which contained 35% to 40% methanol. Although he had been employed in this occupation for four years, his symptoms did not develop until blackout conditions became necessary with a resultant decrease in ventilation. Air analyses at the work place revealed levels from 1.6 to 10.9 mg per liter (approximately 1,200-8,000 ppm). Apparently other employees were not affected, and this employee was considered to have an unusual susceptibility to methanol. His vision improved after exposures were discontinued for six weeks.

Totally, many hundreds of cases of methanol intoxication have been reported as single cases and as groups. In 1951 a mass poisoning occurred with more than 10% mortality when

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320-odd persons were affected as a result of drinking adulterated ethyl alcohol.[10] The symptoms vary with the dose and with the time at which the patient is seen after the ingestion of the alcohol. Early, the patient is inebriated in the same manner as from ethyl alcohol, but not so deeply with an equivalent quantity. Following partial or complete recovery from inebriation, after a delay period of 6 to 24 hours after alcohol ingestion, dyspnea, abdominal pain, vomiting, visual disturbances, delirium, unconsciousness, and coma appear, and death may ensue in several days.

Great confusion has existed about the physiological mechanism of methanol intoxication. A human fatality is reported from as little as 10 cc, while recovery without serious consequences has followed 20 times that dose.[11] It is difficult to accept physiological differences of individuals as a complete explanation of the marked discrepancies reported. The Laboratory for Research in Ophthalmology, Western Reserve University, Cleveland, has reported its experimental results with animals including monkeys in a series of papers entitled "Studies on the Visual Toxicity of Methanol."[12-17] The authors confirmed the work of others by showing that methanol is metabolized differently in primates than in other species, gave a logical explanation of the clinical syndrome and biochemical changes which develop, and proposed a course of treatment for severely intoxicated patients which may be life-saving.

Methyl alcohol is absorbed rapidly from the stomach and intestinal tract and is distributed throughout all body tissues somewhat proportionately to their fluid content. Since it is not metabolized in the brain, the spinal fluid concentration may be greater than the blood concentration.[17] A small percentage is excreted unchanged in the urine and expired air. Methanol is oxidized by the same enzyme that oxidizes ethanol, alcohol dehydrogenase. This oxidation takes place primarily in the liver at one-seventh to one-ninth[18] the rate for ethanol. Since the enzyme is also found in the kidney, some oxidation may take place there. The oxidation products are formaldehyde and formic acid. In the presence of ethanol and methanol, alcohol dehydrogenase oxidizes ethanol preferentially; hence there is competitive inhibition of methanol oxidation when the molar ratio of ethanol: methanol is 1:16, and there is no oxidation of methanol when the alcohols are present in equimolar concentrations.

The marked acidosis which is a prominent feature of methanol poisoning is accounted for only in part by the formaldehyde and formic acid produced. If all the methanol were converted to formic acid, the quantity would be insufficient to cause the acidosis which is observed. It is probable that the metabolites cause secondary enzyme inhibitions so that additional oxidation pathways are blocked, allowing other acids to accumulate and deplete the body alkali.

The Cleveland group explained the syndrome of methanol poisoning as three diseases which could be viewed as three phases of the same disease which occur sequentially: I, narcosis; II, acidosis; and III, nervous tissue destruction.

Disease I results from the acute narcotic action of methanol and is the same manifestation as is seen with other alcohols and solvents capable of causing inebriation and anesthesia. This effect is observed in all animals with doses of approximately 10 gm per kilogram. When methanol and ethanol are consumed simultaneously, as is usual, their narcotic actions are additive.

Disease II is said to occur only in primates and results from the metabolism of methanol to a toxic product, probably formaldehyde. The clinical symptoms are the result of the profound acidosis which is produced. The acidosis exceeds that which would be caused by the metabolism of methanol, and so secondary enzymatic inhibitions must take place. Disease II can be prevented if the presence of ethanol can be maintained for a sufficiently long period of time to allow methanol to be excreted unchanged. Since this phase of methanol poisoning cannot be produced precisely in small animals, and since ethanol is usually consumed before, with, or after methanol, irreconcilable statements about its toxicity have been made in the past.

In monkeys the administration of ethanol prevents acidosis (Disease II) and Disease III. Alkali therapy corrects acidosis but does not completely prevent Disease III, which is central nervous system degeneration with optic nerve and retinal destruction, and basal ganglion necrosis.

That methanol may be harmless when consumed in small amounts with ethanol was shown in a report of 60 cases with a description of 8 patients who were addicted to drinking "methylated spirit," which is ethanol denatured with 4% methanol.[19] Patients may not be seen for treatment until narcosis has passed and acidosis is present. This can be combated by large doses of alkali, the amount required being determined by the carbon dioxide-combining power.

The recommended treatment of methanol

poisoning in the human is based on the foregoing experiments. It consists essentially of providing alkali as needed and ethanol. The tentatively suggested dose of ethanol is 4.5 oz. of 100 proof whiskey for a 70 kg man, followed by 3.0 oz. every four hours. Care must be taken to ensure that the total of both alcohols does not approach 10 gm per kilogram, the acute narcotic dose. Kendal and Ramanathan[20] have suggested that 10 ml of ethanol per hour is near the minimum amount required to suppress completely the metabolism of methanol in human subjects.

Ethanol

Ethanol (ethyl alcohol, grain alcohol, CH_3CH_2OH) is a colorless, flammable, mobile liquid with an aromatic odor. It is completely soluble in water, has a molecular weight of 46.07, and boils at 78.3° C. This alcohol is used extensively as an antiseptic, a tissue preservative, a solvent in pharmaceutical tinctures, and a solvent for a great list of materials found in industry and the home. Its further appearance in the chemical industry is as an intermediate in the production of acetaldehyde, acetic acid, ethyl ether, and esters. Ethanol is made by the fermentation of sugars, forming grain alcohol, and catalytically from ethylene and acetylene. Pure alcohol is hydroscopic and forms a constant boiling mixture with water so that ordinary distillation yields a product containing 95.6% alcohol by weight. Special methods are required to remove the remainder of the water to make 100% or absolute alcohol. Denatured alcohol is ethanol containing adulterants as pyridine, methanol, benzene, and others, which discourage its use as a beverage; and it is exempt from the tax on alcoholic beverages.

The strength of beverage alcohols is measured in proof. Proof spirit (100) in the United States contains 50% alcohol by volume and in Great Britain 49.3% alcohol by weight or 57% by volume. Originally, proof spirit was a liquor containing the minimum amount of alcohol (49.3% by weight) which would catch fire and ignite the gunpowder upon which it was poured for testing.

The American Medical Association Councils on Mental Health and Food and Nutrition have published several reports which explain the current opinions about the metabolism and physiological effects of ethyl alcohol.[21-23] These are summarized here. Water soluble ethanol is absorbed, without digestion, directly and rapidly from the stomach and intestine, the rate of absorption being proportional to the concentration. Following a single dose of 8 ounces of whiskey, the maximum blood concentration is reached in approximately one hour. The absorption rate is slowed if the alcohol is highly diluted, and possibly also if the concentration is sufficiently high to cause mucosal irritation and stimulation of secretions; and it is delayed when food is present in the stomach. Prolonged inhalation of vapors as may occur in industrial employment can cause symptoms; but the amount absorbed through the skin has no practical significance. Alcohol is distributed throughout the body, being more concentrated in tissues of high fluid content. All tissue concentrations are determined by the blood level although there is a lag in the response of tissue levels as the blood level rises and falls, as can be demonstrated by cerebrospinal fluid determinations. Ethyl alcohol is cleared from the body primarily by oxidation. A small quantity is lost in the urine and expired air, usually about 4% with values occasionally as high as 10%. Large doses taken frequently tend to encourage renal and pulmonary elimination. Since bladder urine is a composite, a single urine alcohol determination represents an average for the time of its accumulation and does not measure accurately the blood concentration at any one time. A measurement of the alveolar concentration of alcohol is more representative of the blood level at a given time, and has medicolegal importance. When alveolar and blood concentrations are in equilibrium, approximately 2,000 cc of expired air will contain the same amount of alcohol as 1 cc of blood.

Pharmacologically, ethanol is an anesthetic-narcotic agent with the same depressing actions as other materials so classed. Apparent stimulation results from the suppression of cortical functions, releasing the person from intellectual inhibitions and the lower nerve centers from cortical control. With increasing blood levels there is increasing incoordination and inebriation followed by coma, respiratory failure and finally cardiac failure. Although deaths have occurred at lower levels, a blood concentration of 550 mg per 100 cc is usually fatal if the patient is not treated. This level results from a single dose of about 3 gm per kilogram and represents about 1 pint of whiskey.

Ethyl alcohol is metabolized primarily in the liver by way of the enzyme alcohol dehydrogenase, which is also found to a lesser extent in the kidneys. The oxidation of alcohol to acetaldehyde is relatively slow, whereas the oxidation of acetaldehyde to acetic acid or other acetyl compounds is rather rapid and takes place in many parts of the body. This step can

be delayed or stopped by certain blocking agents, such as disulfiram and butyraldoxime,[24] which allow acetaldehyde to accumulate to toxic levels. When these substances are used deterrently, the unpleasant symptoms of nausea, vomiting, dyspnea, flushing, and palpitation discourage the patient from drinking alcohol. Studies utilizing the isotope C^{14} have shown that more than 90% of ethyl alcohol carbons finally appear in carbon dioxide. The metabolic process, which yields 7 calories per gram, has a maximum rate which is not influenced by the quantity in the body. Estimates of this rate vary, but the rate is about 200 mg per kilogram per hour. A common statement is that man can metabolize 1 oz. of whiskey per hour. The rate may not be the same in all individuals or the same in one individual at different times. When the rate is less than maximum, it can be influenced favorably by the administration of carbohydrate, protein, vitamins, and insulin, all of which have been used therapeutically; but the rate can never exceed the stated maximum.

The terms tolerance, habituation and addiction[21] are often applied to the phenomenon of alcoholism, and sometimes they are used interchangeably. Tolerance is an inherent metabolic quality which determines an individual's basic susceptibility to alcohol. It implies a capacity to consume quantities of alcohol with fewer physiological effects than are experienced on the average. Habituation is tolerance acquired by repeated contacts. One who is habituated shows fewer effects from a given dose than do the uninitiated. This change is not the result of altered absorption, distribution, elimination, or metabolism but seems to be an adaptation of the central nervous system to alcohol. A state of addiction is present when symptoms develop after the withdrawal of alcohol. These symptoms may be of all grades of severity. The dependency upon alcohol from which addiction stems seems to be composed of psychological and physiological components. The person who experiences relief from anxieties and tensions following alcohol consumption is in danger of developing a dependency which can be highly detrimental. Some believe that cells develop a physiological need for alcohol and that withdrawal symptoms appear when this need is not met, an interesting opinion which has not received universal acceptance.

Although ethyl alcohol yields 7 calories per gram, it is a poor nutritional source. When calories from alcohol displace those from conventional foods in the diet, nutritional deficiencies may be expected as a result of deprivation of essential fats, proteins, minerals, and vitamins. The relationship of cirrhosis of the liver to habitual alcohol ingestion has not been absolutely determined. Cirrhosis is found more commonly in those who use alcohol frequently, but it is found also among those who never drink alcoholic beverages. The liver, being the main organ for the detoxification of alcohol, may suffer from its direct toxic effect or the hepatic changes may be secondary to nutritional deficiencies. Although alcoholism has been studied extensively, many facets of this complex disease require elucidation.

Isopropanol

Isopropanol (isopropyl alcohol, 2-propanol, dimethyl carbinol, $CH_3CHOHCH_3$) is a colorless, flammable liquid with an odor similar to but less sharp than ethanol. It has a molecular weight of 60.10, boils at 82.3°C, is completely soluble in water, and upon drying from the skin leaves a slight oil-like feeling. It has solvent properties like those of ethanol, which it has displaced in some industrial uses, for, since isopropanol is not suitable for drinking, its distribution and use in commerce are not hampered by the legal restrictions that regulate beverage alcohol. A common household product is isopropyl alcohol rubbing compound which is about 70% isopropanol in water. A concentration of 30%-50% of this alcohol in water is used as an antiseptic. Several commercial methods are available for its production, but most is manufactured by the reaction of propylene with sulfuric acid to form isopropyl sulfate followed by hydrolysis and recovery of the alcohol and acid.

Isopropanol is absorbed rapidly from the intestinal tract and the lung and is distributed throughout the body. It is oxidized slowly to acetone in the liver. Acetone also is an effective narcotic with approximately the same toxicity as isopropanol (LD_{50}'s 9.75 and 10.7 gms per kilogram, respectively) and may contribute to the narcosis from isopropyl alcohol. The alcohol is cleared from the body unchanged via the lung and kidney and by oxidation to acetone, which may be excreted or metabolized further.[25]

Isopropanol is said to be twice as narcotic[26] and twice as toxic[27] as ethanol. When the LD_{50} of ethanol, 21.3 gm per kilogram, is compared with that of isopropanol, 10.7, and when the estimated, approximate, human fatal doses are compared, 250 cc and 160 cc, respectively, the statement about toxicity seems to be correct. However, when comparisons are made of the blood alcohol levels of severely intoxicated patients, 130 mg[28] to 150 mg[29] per 100 cc for isopropanol and 450 mg to 550 mg for ethanol,

it would appear that the narcotic effect of isopropanol is approximately three times that of ethanol. In one case acetonuria was reported, and the odor on the breath resembled acetone; so acetone may add to isopropanol narcosis.

Human intoxications have occurred from the ingestion of isopropanol[29] and following its use in large quantities (12 to 20 oz.) in alcohol sponging of febrile children in confined, poorly-ventilated areas.[28, 30] It was thought that these intoxications in children resulted from the inhalation of the alcohol. One child became unconscious in four and one-half hours and another was found in a coma nine hours after being wrapped in a towel saturated with the alcohol. The patients present unconsciousness, deep breathing or rapid, shallow breathing, rapid pulse, and constricted pupils which do not respond to light. With supportive treatment, recovery can be expected in 24 hours.

Butanol

Butanol (*n*-butanol, butyl alcohol, CH_3CH_2-CH_2CHOH) is a colorless mobile liquid with pungent odor. It has a molecular weight of 74.12, boils at 117.7°C, and is soluble in water to about 7%. It can be made by the fermentation of sugars or synthetically from propylene or acetaldehyde by several production steps. Being an excellent solvent, it is used in the manufacture of resins, varnishes, lacquers, and related substances.

Butanol is absorbed from the intestinal tract and pulmonary surfaces rapidly and is metabolized completely by oxidation.[25] Its primary physiological effect is narcosis, being more active than the lower numbers of the series. The LD_{50}, 4.3 gm per kilogram, is smaller than those for the lighter members, indicating greater toxicity. The vapors can cause irritation of the conjunctiva. The concentration at which this occurs has been reported as 50 ppm[31, 32] and 200 ppm.[33] Conjunctivitis and keratitis with pain and disturbance of vision associated with the presence of intracorneal vacuoles have been reported in workers exposed to *n*-butanol[34] and to a mixture of butyl acetate and isobutyl alcohol.[35] A similar condition described as "fine punctated turbidity of the superficial layer of the cornea" which may represent the same pathology followed exposure to butyl acetate and butyl alcohol.[25]

A peculiar antihemorrhagic action of *n*-butanol has been observed.[36] When the alcohol was given orally, intramuscularly, or intravenously in doses of 5 to 10 cc, active bleeding was stopped in two to three minutes in 24 of 25 patients with far advanced cancer. The authors concluded that the beneficial effect was related to the action of butanol on the metabolism of abnormal cells rather than on the clotting mechanism.

Welt[37] reported the use of *n*-butanol as a 7.9% solution in saline for the control of postoperative pain in otolaryngological surgery. Doses of 5 to 10 cc for adults and 0.5 cc per year of age for children relieved pain in 90% of the 938 patients treated. Following the intramuscular injection of the dose, relief from pain at times was observed within 5 minutes and usually within 15 minutes. No ill effects were found after the intravenous administration of 500 cc of a 7% solution of the alcohol in saline. Welt also reported that Ravich and Revici had given patients oral doses of 1,500 cc daily for 10 days without any immediate or delayed undesirable side effects.

Summary and Conclusions

The saturated, open-chain, monohydric alcohols have been reviewed in reference to their general actions and chemistry. As a group they are anesthetic-narcotic agents similar to the other organic solvents, and by some measurements the intensity of this action increases progressively with the chain length. Among isomers, branching and locating the hydroxy radical in secondary and tertiary positions decrease the narcotic action when comparison is made with the normal compound. The important individual alcohols up to butanol are discussed in some detail. Although reports of serious methanol intoxication are no longer received from industry, it remains a potentially hazardous chemical. The absence of industrial intoxications from the alcohols reflects the low order of toxicity of this homologous series and indicates that adequate environmental control over these solvents is maintained.

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Diagnosis and Compensation of Radiation Injury

Statement on the Diagnosis and Compensation of Harmful Effects Arising as a Result of Work Involving Exposure to Ionizing Radiation

JOINT COMMITTEE REPORT

ON THE basis of the best knowledge presently available, the Radiation and Workmen's Compensation Committees of the Industrial Medical Association recommend the following basic guides for the diagnosis and compensation of harmful effects arising as a result of work involving exposure to radiation.

Need for a Statement

Many of the difficulties encountered in compensation for injury possibly resulting from exposure to ionizing radiation at work are similar to the problems encountered in compensating for other occupational injuries. However, certain features of radiation injury introduce diagnostic problems which complicate the administration of compensation. In order to avoid misunderstanding and misinterpretation the following statement is presented.

Many members of these two committees are members of other committees working on this problem. The work of one such committee, that appointed by the Atomic Energy Commission, has been drawn upon heavily in the preparation of this statement because of the collective experience of this group in the actual handling of radiation injuries (Impairment of Health by Ionizing Radiation: Report submitted to the Joint Committee on Atomic Energy for inclusion in proceedings of the Hearings on Employee Radiation Hazard and Workmen's Compensation, March, 1959).

Considerations in Establishing Causal Relationship Between Occupational Exposure to Radiation and Injury from Such Exposure

Much has been learned since the turn of the century about the effects of ionizing radiation on animals and man. This knowledge has been accumulated through many observations on man and extensive experimentation with laboratory animals. It is now becoming generally recognized that the effects of ionizing radiation have been studied more thoroughly than the effects of many other environmental agents capable of affecting health. Much is known about the effects of large doses of radiation rapidly administered. These well-known effects, such as the acute radiation syndrome, which result from exposure of all or most of the body to a large rapidly administered dose of penetrating radiation will not be discussed, since they are covered by a large and generally noncontroversial literature. ([*a*] Thoma, G. E., Jr. and Wald, N.: The Diagnosis and Management of Accidental Radiation Injury, *J. Occup. Med.* 1:421 [Aug.] 1959; [*b*] Norwood, W. D.: Skin Hazards from Radiation in Atomic Industry, in Occupational Diseases of the Skin, Swartz, L., Tulipan, L., and Birmingham, D. J., Philadelphia, Lea & Febiger, 1957, Chap. 2, Medicine and Defense). Less is known about the effects of very small doses. While there are some gaps in our knowledge, current information is sufficiently comprehensive to provide a basis for the diagnosis and compensation of radiation injury.

A. General factors to be considered in establishing a diagnosis of injury due to exposure to ionizing radiation in industry.

1. There are many sources of radiation exposure. In addition to possible exposure in multiple work places, employees are constantly exposed to natural radiation and to the less significant radiation from atomic fallout. They may also receive significant exposure from diagnostic and/or therapeutic radiation. Miscellaneous other sources may also contribute to radiation exposure. The report of the United Nations Scientific Committee on the Effect of Atomic Radiation, New York, 1958, states, "at present, the exposure to ionizing radiation received occupationally forms only a small contribution to the total irradiation of the population as a whole, amounting to about two per cent of that from natural sources in countries in which occupational exposure is probably largest." This is important when considering genetic damage. It should be

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remembered that individual exposure is of primary concern in determining somatic injury.

2. Most diseases and injuries, including delayed conditions, which may be induced by radiation may also result from other, often unidentifiable, causes.
3. There may be a long latent period between the time of exposure to radiation and the onset of symptoms.
4. Many radiation effects occur only when definite minimum or threshold exposures are exceeded. Based upon animal experiments, most geneticists feel that any exposure to ionizing radiation will produce genetic mutations. The frequency of such mutations appears to be dependent on dose rate (Russell, W. L.; Russell, L. B.; and Kelly, E. M.: Radiation Dose Rate and Mutation Frequency, *Science* 128, Dec. 19, 1958). Whether the statistical probability of the occurrence of such conditions as leukemia, cancer and life shortening is proportionally or otherwise increased by very small doses or whether there is a threshold dose below which these effects do not occur cannot be answered at present.
5. The relationship of specific somatic effects to dose may vary depending upon age, sex and constitutional differences. Fetuses, infants and children are more susceptible than adults for certain effects.
6. The effect of external radiation exposure will vary depending upon: (*a*) absorbed dose; (*b*) dose rate; (*c*) distribution of dose in time (continuous over period or intermittent) and space (within organs and tissues); (*d*) extent of exposure—whole body or partial body; (*e*) sensitivity to radiation of tissue or system exposed; (*f*) biological effectiveness of the radiation for the tissue considered; (*g*) the type and energy of the radiation; and (*h*) nature of effect being considered.
7. The effect of a radioisotope deposited in the body will vary depending upon (*a*) the site of deposition in organs; (*b*) the half-life of its radiation and time of retention in the organ; (*c*) the type and energy of the radiation emitted; (*d*) the sensitivity of the critical organ or tissue to radiation injury; and (*e*) essentiality of organs affected. Detailed information concerning the physical characteristics and measurement of radiation, critical organs, recommended maximum permissible exposures, etc., may be obtained from handbooks of the National Committee on Radiation Protection and Measurements published by the National Bureau of Standards, Washington, D.C.
8. Exposure to relatively large doses of radiation, rapidly administered, produces a variety of characteristic effects which may occur within minutes or may be delayed for days and years.

B. More specific considerations in diagnosing occupational injury from radiation.

1. Physiological changes due to radiation. As in the case with many other environmental factors affecting human health, transient physiological, biochemical and histological changes may be caused by ionizing radiation. Such changes may include, for example, shifts in the mitotic index in various tissues, changes in the differential and total white blood counts, an increased incidence of bilobed or binucleated lymphocytes and an increase of desoxyribonucleic acid in lymphocytes. Such findings, in the absence of future evidence to the contrary, should not be construed as evidence of harmful effect.
2. Relationship of harmful effects to exposure. The Report of the United Nations Scientific Committee on the Effects of Atomic Radiation (A 3838, New York, 1958) concludes: "In human adults it is difficult to detect the effects of a single exposure to less than 25 to 50 rem, or of continuing exposure to levels 100 times the natural levels."

   At levels far above the presently accepted "maximum permissible exposures," ionizing radiation will by itself cause recognizable harmful effects. The National Committee on Radiation Protection and Measurements (NCRP), defines harmful effect as "any body injury, disease, or impairment, except where such condition is transitory, infrequent, or of short duration and does not endanger persons so affected" (National Bureau of Standards Handbook 61).
3. Assessment of harm. The degree of harm to health in each case can best be determined by physicians skilled in the field of ionizing radiation injury. The Committee on Medical Rating of Physical Impairment of the American Medical Association states: "Evaluation of permanent impairment defines the scope of medical responsibility and, therefore, represents the physician's role in the evaluation of permanent disability. Evaluation of permanent impairment is an appraisal of the nature and extent of the patient's illness or injury as it affects his personal efficiency in the activities of daily living" (*Journal of the American Medical Association*, Special Edition, Feb. 15, 1958). The statements in this Section B are largely derived from the Report to the Joint Committee on Impairment of Health by Ionizing Radiation, referred to previously.

C. Considerations involving diagnosis of and com-

sation for long delayed effects of ionizing radiation.

1. Tissue repair—threshold effect. Processes of repair greatly influence the final outcome of radiation damage in the individual. They are one reason for the existence of the threshold dose or dose rate effect. The minimal or threshold dose or dose rate must be exceeded before an observable biological effect occurs. A familiar example of this phenomenon is the redness of skin which occurs only when a minimal dose or dose rate of radiation is exceeded.
2. Damage dependence on dose rate. For most effects it is obvious that injury is much less at low dose rates. For example, in the past, radiologists were exposed, over a period of many years, to a cumulative dose which if administered in a short period of time (e.g., 30 minutes) would have caused death in days or months. The only significant measurable evidence of injury from such exposure to date has been a slight statistical increase in the incidence of leukemia. Other aspects of the subject will be considered in discussing specific types of delayed or late effects.
3. Genetic damage. Fifty million fruit flies were required to demonstrate that, with rapidly administered radiation doses in the range of 25 to 50 r, genetic damage was roughly proportional to the dose (Spencer, W. P., and Stern, C.: *Genetics* 33:43-74 [Jan.] 1948). Based on this and work with mice, it is generally inferred that any dose of radiation will increase the statistical probability of genetic damage in proportion to the dose. However, recent experimental work with mice indicates that the mutagenic effectiveness of a protracted dose of gamma radiation is much less than that of the same dose administered rapidly as a single dose (Russell et al., previous reference). Extrapolation from fruit flies to man and from doses of 25 r of rapidly administered radiation to doses of 0.1 r slowly administered may introduce errors of considerable magnitude. This indicates a need to extend experimental work to the lowest practicable levels, and to elucidate further the fundamental mechanisms of biological action.

   Even though mutations may be induced in an individual, identifiable damage to his traceable offspring may never occur. If damage does result it may be delayed for many generations. Industrial ionizing radiation accounts for only a small fraction of the total radiation exposure of the population and, therefore, for only a small fraction of the total load of mutations in that population. Therefore, the statistical chance that a particular genetic abnormality in an offspring has resulted from occupational exposure of a parent would be small and it would be very difficult to relate such damage to specific exposure. If human genes have the same response to radiation exposure as do those of mice some 40 to 240 rems would be required by each generation to double the present mutation rate for the population of the country. The large range is due to the dose rate effect demonstrated by the Russells and previously referred to. "A doubling of the mutation rate might lead to an increase of one in a thousand (0.1%) in the numbers of harmfully affected children in the next generation" due to dominant-type gene damage (The Hazards to Man of Nuclear and Allied Radiations, Report of Medical Research Council, London, Her Majesty's Stationery Office, June, 1956). On this basis, an employee receiving an acute dose of say 400 rems would receive $\frac{400}{40}$, or ten times as much genetic injury as if he had received the doubling dose. The probability of harmful genetic damage appearing in the immediate offspring, as a result of such an exposure would be $10 \times 0.1\% = 1\%$. Since only one parent would probably receive the excessive exposure the 1 per cent might be reduced to one-half per cent. If the 400 rems were accumulated slowly over a lifetime the probability of harmful genetic damage would be reduced by a factor of about four or the chance would be about one in 800 ($\frac{1}{8}\%$). Mutations of the so-called recessive type might have some effect on first generation offsprings but recessive effects would largely be spread over an indefinite number of generations. When an abnormality, considered to be of genetic origin, appears in the immediate offspring of a worker having a high radiation dose from occupational and other exposure the above information may be used in estimating the statistical chance that the abnormality is due to the occupational exposure.
4. Life shortening. Exposure of laboratory animals to large, rapidly administered, sublethal doses of radiation has resulted in statistical life shortening which is roughly proportional to the dose. However, the duration of life of animals receiving less than 10 rems per week has not been significantly different from that of controls which received no exposure. (Mole, R. H.: Shortening of Life by Chronic Irradiation—the Experimental Facts, Report of the United Nations Scientific Committee on the Effects of Atomic

Radiation, New York, 1958, p. 153.) Radiologists, exposed in the past in this country and in England to doses possibly as high as 2,000 r over a period of about 20 years, have not been shown to have any statistically significant life shortening. Therefore, it seems reasonable to infer that chronic industrial exposures many times greater than the maximum permissible, would probably not cause appreciable life shortening. Acute whole-body doses in excess of 200 rems might result in detectable life shortening, if man reacts in this respect in a manner similiar to laboratory animals. This would be detectable only on a statistical basis in a large group, and not in an individual case. Because of the multiple factors which determine the length of life of an individual, the relative contribution of a sublethal dose of radiation to life shortening would be extremely difficult to determine. Such factors as smoking a pack of cigarettes daily, excess weight, or improper diet might have a far greater effect on longevity than such a dose of radiation. Each case would have to be individually considered in light of subsequent impairment which might be related to exposure and might influence longevity.

5. Leukemia. The existence of a radiation threshold for the causation of this uncommon disease is a highly controversial issue. Animal studies and observance of the incidence of leukemia in radiologists, among Hiroshima and Nagasaki survivors and in children irradiated in utero during radiographic examination of their mothers, indicate that radiation at high dose rates increases the occurrence of leukemia in the exposed groups. In American radiologists, the doses which resulted in a statistically significant increase in the incidence of leukemia were in the order of 100 r per year (Braestrup, C. B., *Am. J. Roentgenol.* 98, Dec. 1957). Administration was rapid but in small and frequently repeated doses. The effectiveness of chronic exposure to low dose rates is less clear. Evaluation of the possible role of industrial exposure in the causation of leukemia—particularly at or below the maximum permissible exposure rates—will require extremely painstaking study by the best qualified experts.
6. Malignant disease other than leukemia. The production of malignancies by radiation has been known since cancer of the skin appeared in the early days of medical application of x-rays. Sarcomata (and bone necrosis and aplastic anemia) have occurred in radium workers, due to deposition of this radioisotope in bone. The determination of the probability that a given malignant disease is or is not due to occupational exposure to radiation does not differ basically from the situation in which an industrial chemical is the suspect carcinogen and thus should present no greater difficulties than those already being solved.
7. Cataracts. It is known that cataracts may be caused by ionizing radiation. The minimum dose of x-rays to produce cataracts in humans has been determined with a fair degree of accuracy by measuring the exposure to the lens when a malignant disease of the eye has been treated with measured doses of roentgen rays. The smallest dose which has caused cataract in humans to date is about 200 rems. Much larger doses have been given without producing cataracts. If the exposed persons are observed over an appropriate period, the specially trained ophthalmologist may sometimes be able to differentiate between the radiation induced cataract and that due to other causes by the appearance and sequence of changes in the lens.
8. Causal relationship in specific disease. In addition to the more commonly used clinical means, statistical methods will be increasingly useful in determining the degree of probability of a causal relationship between occupational exposure to radiation and late effects such as the development of leukemia or other malignant disease. To this end it is most important to maintain adequate cumulative records of employees' exposure to external and/or internal radiation.

   When investigating a case of delayed illness or injury possibly caused by occupational exposure to radiation, physicians with experience in the field of ionizing radiation injury should correlate all available exposure information with clinical evidence of possible radiation injury. They should consider all of the factors previously outlined in establishing a diagnosis of injury due to exposure to radiation in industry. After a thorough investigation of all the facts, physicians with experience in the field of ionizing radiation are best qualified to render an opinion concerning the existence and degree of impairment and whether the probability is greater or less than fifty per cent that the impairment is the result of exposure to ionizing radiation at work. The opinion of such physicians should be given great weight by those responsible for arriving at a decision as to whether an employee with illness or injury should receive compensation and, if so, what the amount of the compensation should be.
9. Modification of state compensation laws.

It is recognized that some changes are necessary in the compensation laws of some of the states to make them adequate to handle the special problems of radiation injury.

The International Association of Industrial Accident Boards and Commission (IAIABC) have made recommendations, including the following, which are listed in abbreviated form with one exception. The recommendation for "making radiation cases, upon establishment of multiple exposure, eligible for second injury fund benefits," was not included. While our committee endorses this principle of utilizing second injury funds, we doubt the practicability, due to difficulty in establishment of prior injury. (Selected Materials on Employee Radiation Hazards and Workmen's Compensation, Joint Committee on Atomic Energy, Congress of U. S., February, 1959.)

a. All State Workmen's Compensation laws should cover occupational diseases as well as industrial accidents. Mississippi and Wyoming did not provide disease coverage as of July 31, 1959.
b. Occupational disease laws should include provision for compensation for radiation injury. Based upon the U. S. Department of Labor survey and subsequent changes, there appear to be five states which did not cover injuries due to radiation as of July 31, 1959. These are Alabama, Mississippi, Montana, New Hampshire and Wyoming.
c. Because of the possibility of delayed effects of radiation, the time limitations in filing claims should begin to run from the date on which the employee has knowledge of the nature of the disability and its relation to his job.

The Radiation and Compensation Committees urge that every effort be made to encourage states to enact laws when necessary to comply with these recommendations.

10. Precautionary work changes. The work of employees may be changed for varying periods of time due to exposures which have not caused injury but which exceed the maximum permissible limits. Such changes in work could conceivably result in loss of earnings but this should be clearly differentiated from losses resulting from overexposure with resulting injury.

Conclusion

While the diagnosis and compensation of radiation injury in industry is not easy, it is felt that it may be accomplished in an equitable manner on the basis of information now available and within the general framework of current compensation practices.

It is hoped that other medical groups interested in establishing criteria for the diagnosis and compensation of harmful effects, arising out of occupational exposure to radiation, will add their efforts to those of the Industrial Medical Association in producing criteria for consideration of the American Medical Association. This statement should be reviewed periodically and modified when necessary as new data become available.

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TWO POSTGRADUATE COURSES ON DISEASES OF THE CHEST

The 15th annual course, "Clinical Cardiopulmonary Physiology," will be held at the Sheraton Towers Hotel in Chicago, October 24-28, 1960. Co-chairmen are Albert H. Andrews, M.D., Associate Clinical Professor of Bronchoesophagology, University of Illinois College of Medicine, and Edwin R. Levine, M.D., Assistant Professor of Clinical Medicine, Chicago Medical School.

The 12th annual course, "Recent Advances in the Diagnosis and Treatment of Diseases of the Heart and Lungs" will be held at the Park Sheraton Hotel, New York City, November 14-18, 1960. Co-chairmen are Edgar Mayer, M.D., Clinical Professor of Medicine, New York University Postgraduate Medical Center; Alfred S. Dooneief, Lecturer in Medicine, Columbia University College of Physicians and Surgeons, and Emil A. Naclerio, M.D., Chief Thoracic Surgical Services, Harlem and Columbus Hospitals, New York City.

Tuition for each five-day course will be $100 including round table luncheon discussions. Additional information may be obtained by writing to the American College of Chest Physicians, 112 E. Chestnut St., Chicago 11, Ill.

Industrial Medical Association

THE PRESIDENT'S MESSAGE

ROBERT E. ECKARDT, M.D.

ONE OF THE difficulties in the past with the annual IMA meeting has resulted from the lack of continuity in the Scientific Program Committee. Precedent has established that the chairman and members of the Program Committee would all come from the locale in which the annual meeting was to be held. The result has been repetition of subjects, omission of subjects, and generally a multitude of complaints including, on the one hand, that our annual scientific program is directed primarily at the full-time industrial physician and overlooks the needs of the part-time man or, on the other hand, that it is so simple and fundamental that it has no interest to the full-time man. Your Executive Committee has suffered patiently through these complaints each year, each member of the Executive Committee having had past painful experience with the planning of an annual meeting. After due consideration, the Executive Committee has decided to institute a new system for the Scientific Program Committee for our annual meeting. The Chairman of this Committee will still be selected from the locale of the annual meeting, but to assist him will be the Chairmen of the last two Scientific Program Committees, and the two Chairmen of the coming meetings.

Thus for our 1961 meeting, Dr. J. S. Felton of Los Angeles will be Chairman of the Program Committee, and will be assisted by Dr. Eugene L. Walsh of Chicago (1959 Chairman), Dr. Harry A. Hanson (1960 Chairman) and Dr. Gilbeart H. Collings Jr. (1962 Chairman for Chicago). Since the exact site of the 1963 meeting has not yet been selected, the fifth member of the Committee has not yet been selected. As time progresses, each succeeding Chairman will have had the benefit of at least two years' experience on the Committee, and thus, it is hoped, will bring a continuity of program planning to our annual meeting which has been absent in the past. It is hoped that this change, along with the designation of the Chairmen of our Scientific Committees as an informal Program Advisory Group will do much in the future to improve the quality of our scientific program, as well as to broaden its interest to all our members. Every member is invited to send to our Program Committee any suggestions they may have for program content for this or succeeding years. Help your Program Committee to make your annual meeting what you want it to be!

In Los Angeles in 1961, we are planning also to have concurrent sessons dealing with toxicology, radiation, and industrial hygiene, since the AIHA and ACGIH will not be with us. It is hoped that these will be a practical level to attract and interest our physician group.

It is planned also to organize our Scientific Exhibits Committee according to the format of the Program Committee, with the Chairman of the present meeting being assisted by the Chairmen of the immediate past meeting and the next future meeting. In this way we hope to bring you better and more interesting scientific exhibits.

Finally, the IMA Public Relations Committee will be given responsibility for IMA publicity prior to and during our annual IMA meeting. In each case one member of the Committee will come from the local area of the annual meeting in order to establish the local contacts necessary for publicity outlets.

Your Executive Committee believes that these changes are important ones for the continuing success and improvement of our annual meeting. It is hoped that in this way our annual meeting will become, even more than it is now,

the most outstanding industrial medical meeting in the world. We hope that our annual meeting will attract, and be of interest to, almost 50% of our membership, rather than the 17% who have attended past meetings. Broadening the base of participation in association affairs should lead to a bigger and *better* IMA.

Book Reviews

LUMBAR DISC LESIONS: PATHOGENESIS AND TREATMENT OF LOW BACK PAIN AND SCIATICA. J. R. Armstrong, M.D., M.Ch., F.R.C.S. Ed. 2. Price, $12.00. Pp. 244. The Williams & Wilkins Co., Mt. Royal & Guilford Aves., Baltimore 2, Md., 1958.

In an era of increasing awareness of degenerative changes in the lumbar discs as the major cause of low-back pain, with or without sciatica, Armstrong's second edition provides a sound, readable, and well-documented summary of current views of British orthopaedic surgeons on the low-back problem. Although titled "Lumbar Disc Lesions," the sections on differential diagnosis include such complete descriptions of other causes of low-back pain that the monograph actually represents an excellent modern text on back-ache.

The book is intensely practical. Clinical descriptions are complete and are derived from clearly delineated anatomical and pathophysiological concepts. Examination techniques are fully explained and illustrated. Details of conservative and operative treatment are presented in didactic and authoritative fashion.

Striving for clarity and completeness, the text occasionally becomes repetitious and one has the feeling that as much material could be covered in better organized form in 50 fewer pages.

The author makes several important points with telling effectiveness. The section dealing with the relationship between trauma and disc damage will be of extreme interest to all who deal with the industrial low back. Myofascial strains, sacroiliac and lumbosacral sprains and postural strains are put firmly and deftly into their proper places as sources of low back pain.

Paralleling growing trends of thought in this country, Armstrong doubts the value of myelography in the diagnosis and localization of lesions of the lower lumbar discs, pointing to false positive and false negative results in more than 20% of cases. He decries discography as unjustifiable.

The section on conservative treatment will provoke minor disagreements in this country. Apparently, the lumbar flexion position is not so highly regarded in Britain as in America. The emphasis on the importance and over-all efficacy of conservative treatment, however, is well underlined.

Operative treatment is illustrated by excellent color sketches. The author favors a wider approach with removal of more bone than is currently favored in this country. Although recognizing the theoretical advantages of fusion, he pinpoints clearly the practical difficulties of obtaining solid fusions in disc disease and concludes opposed to the routine or even frequent performance of the combined disc-fusion procedure.

The book is well laid out. Although not copiously illustrated, the sketches are clear and meaningful and the x-ray reproductions are superb. The text material is effectively presented, and reads easily. Although didactic, it is never overbearing.

In the opinion of this reviewer, the book represents the best summary of modern thought on the low-back problem currently available.

—M. LAURENS ROWE, M.D.
University of Rochester School of Medicine and Dentistry
Rochester, N. Y.

ASSESSMENT OF HUMAN MOTIVES. Sponsored by the Department of Psychology, Syracuse University, edited by Gardner Lindzey, University of Minnesota. Price, $5.00. Pp. 273, with no illustrations. Rinehart & Co., Inc., 232 Madison Ave., New York 16, N. Y., 1958.

The title of this book suggests that it might be relevant to problems in human motivation which impinge on occupational medicine. The assessment of human motives can play an important part in dealing with problems such as absenteeism, accidents, prolongation of disability, and compensation claims. The industrial physician, however, would find this book difficult to read and not immediately rewarding, in terms of his background and experience, to the assessment of motivations which complicate industrial health activities.

Actually the book does not claim to present practical applications in any particular sphere of human endeavor. It presents nine theoretical and methodological approaches to the experimental study of the psychology of motivation. This is an aspect of psychology in which there has been a considerable gap between the objective experimentalists and the more intuitive clinicians. The approaches in this book offer various possibilities toward reducing this gap and furthering the scientific examination of the understanding, prediction, and modification of behavior, without neglecting the individual as he is influenced by both biological and sociological factors, through conscious and unconscious routes. They offer several alternatives of basic importance for research in psychiatry, clinical psychology, and industrial psychology.

Although this is not a book to be recommended to most readers of this journal, it is one that can be expected to influence the course of research which will some day find application in the mental health aspects of occupational medicine.

—W. DONALD ROSS, M.D.
Department of Psychiatry
Cincinnati General Hospital
Cincinnati 29, Ohio

ELEMENTARY STATISTICS WITH APPLICATION IN MEDICINE AND THE BIOLOGICAL SCIENCES. Frederick E. Croxton. Paper. Price, $1.95. Dover Publications, Inc., 180 Varick St., New York 14, N. Y., 1959.

The present volume maintains the same conciseness of presentation and wealth of materials contained in the earlier, hard cover edition of Croxton's "Elementary Statistics with Applications in Medicine." It requires a modest knowledge of mathematics and may serve as a convenient reference for elementary problems as well as a text for introductory or refresher course.

The first five chapters deal with the usual descriptive materials. Little if any emphasis is placed on preparation of the reader for the concepts introduced at a later point. Regression, correlation, and linear and multiple correlations are introduced before discussion of distributions and tests between means. Such sequencing in fairly unusual. Despite the difficulties inherent in comprehending the subject presented in that particular order, it also implies an emphasis on estimation statistics, often the more valuable uses of this tool for biological research. In fact, the normal emphasis on so-called tests of significance may have done much toward influencing research design into less efficient channels.

The presentation of nonlinear regressions has an elegance of simplicity and clarity that would be difficult to match. However, one can also note that the change in title from the previous volume has not been paralleled by appropriate changes in the material. Some purely biological topics which would have fitted well into the discussion at that point have not been added. Most notably among the missing are techniques of bioassays.

There is a good discussion of distributions which does mention the Poisson. In view of proliferation of research involving radioactive materials it is regretful that the discussion deletes references to applications of the Poisson. Nor are radiological statistics mentioned. Other and more routine tests are ably presented, with fitting examples.

For its size, and certainly for its price, the volume offers much in practical value. The run-of-the-mill biologist with the usual preparation of one introductory course should make good use of the material given him. Useful to him, too, will be the dictionary of symbols that prefaces each chapter. It will make it easier for him to follow examples and development of arguments. Well-placed section and paragraph headings will be of help too. Despite all this, there is little about the work that seems to be especially pertinent to medical or biological research. It's just a good introductory text and no less desirable for that.

—THEODOR D. STERLING, Ph.D.
Director, Department of Biometrics
Department of Preventive Medicine
and Public Health
University of Cincinnati
Cincinnati, Ohio

Information Please

The questions published in this column are edited versions of requests for information that have been received during recent months. The answers have been prepared by one or more competent authorities in the particular area covered by the question. Readers may address questions to the Editor.

QUESTION: *Does 33% sodium hydroxide solution (when used for cleaning grease from the floor) produce a mist that irritates the eyes and mucous membranes of the upper respiratory tracts of workers so exposed?*

When the solution is cold, there should be no mist from 33% sodium hydroxide. However, when a solution of that concentration is used in contact with grease and fats, it is possible that the heat of reaction (during saponification) could produce a mist that would be irritating to mucous membranes.

QUESTION: *What is the best way to handle 33% sodium hydroxide solution on open cleaning jobs?*

If such a concentration (33%) of sodium hydroxide must be used, it is recommended that exposed persons wear rubber boots, rubber aprons or raincoats, rubber gloves, and chemical type goggles. If a 10% solution of sodium hydroxide will serve to clean the floor, irritation of mucous membranes is less likely to be a problem. It is understood that floor cleaning in the paint, varnish, and lacquer industry is done with 10% sodium hydroxide.

The following alternate method of cleaning may prove useful: A preliminary steaming is done to loosen the grease so that it can be flushed away, and then a mild alkali is applied to whatever grease remains. The use of a phosphate cleaner should also be considered. A respirator and protective clothing is required regardless of the cleaning method that is employed. A relatively safe, nonflamable solvent, such as trichlorethylene, may be used where it is possible to provide local exhaust ventilation that will ensure against the inhalation of vapors above the maximum allowable concentration.

Editorial

The Lion and the Lamb

THE LEAD articles in this issue deal with an industrial medical program and a joint union-management health program in the same industry: the hotels of New York City. Having long wondered what relationships might exist in such a situation, we recently went to find out.

For legal purposes the hotel industry's Health Center carries a long name that denotes its sponsorship by union and management: New York Hotel Trades Council and Hotel Association of New York City Health Center, Incorporated. It is the business of the Hotel Health Center to provide general medical care to employees at management expense, an arrangement arrived at by collective bargaining. Whether this is the best way to solve a medical care problem is beside the point. Our present question is, how does such a plan work alongside an occupational medicine program provided by management at the place of employment?

A quick answer—with all the hazards of a quick answer—is that it appears to work reasonably well in the present situation. The boundary line between the two types of service is a fluid one. Where no employee health service is provided within a hotel, the employer-maintained Health Center is used extensively; where good in-hotel medical service for employees exists, the Center is limited to nonoccupational medical care.

The performance of preemployment examinations at the Health Center seems to create no problems. The jobs of the industry are not associated with any recognized occupational disease hazard, and the objective of the preemployment examination is limited to a careful grading of the prospective employee with respect to the job under consideration. In this regard the interests of the Center and the prospective employer are in harmony. The employer wants productive hands and the Health Center prefers future patients who are average-or-better medical care risks. Were there complicated Workmen's Compensation Law problems or were the cost of general illness immediately chargeable to the employer instead of being lost in a common assessment over which he has virtually no control, one suspects that there might be some contest for the direction of preemployment as well as the periodic health examination functions.

The Hotel Health Center draws a sharp line when it comes to treating occupational conditions. These cases are sent elsewhere—to the employer's medical department, if he has one, or to the physician designated by the insurance carrier concerned.

One readily believes that the Health Center provides a great convenience to hotel physicians and nurses who wish to make good dispositions of nonoccupational injury and disease cases among employees. There at the Health Center, under one roof, all medical specialties are represented and utilized, and competent people are available to handle the paperwork of complicated social insurance and public welfare programs; and a system exists for providing home and hospital care. What might otherwise be a full afternoon's work for an interested hotel nurse determined to solve a medical care problem for an employee is now accomplished quickly to the apparent satisfaction of all concerned.

A tempting speculation here is that the traditional adversaries, union and management, in this instance have found a noncontroversial area of common interest in which the unique resources of each camp have been joined to solve a broad community problem in better fashion than would be possible were either to try it alone.

Abstracts of Current Literature

Medical Care Administration

Management's Interests in Medical Care Plans. S. W. Wirpel.* Monthly Labor Rev. 83:147 (Feb.) 1960. (*Employee Benefits, Inland Steel Company Chicago, Ill.)

There is no essential difference between labor's view and that of management as to their responsibility for medical care. They differ as to a question of costs and willingness to sacrifice money wages to pay for the desired level of medical care.

Both management and labor must gear their programs to meet the needs of the community. A limited group-practice or open panel plan could operate on about $200 per employee per year: at the present in most industries about $100 per employee is available. The company and union should agree to contribute an amount in lieu of a wage increase for one, two, or three years until sufficient capital is available to acquire needed facilities. A framework should be developed for a community plan open to all groups and individuals in the community. An annual choice of participation or nonparticipation of group practice or open panel or a limited traditional type of hospital-surgical insurance should be permitted. A community approach is needed, not a nationwide union-management plan limited to this or that union or industry. —A.B.

How the Medical Profession Looks to the Layman. J. F. Allen.* California Med. 91:344 (Dec.) 1959. (*559 Greer Road, Palo Alto, Calif.)

Most people sharply distrust medicine as a profession: they are, however, usually lost in a sort of magic worship of any doctor who treats them. This is the observation of the author, a San Francisco journalist. Doctors, like many other groups, tend to sit around and sell each other on ideas already widely held by the group, then assume that everyone thinks that way, too. They are blind to the fact that they are leading a crusade without a following. Such phrases as "free choice of physician," "third party medicine," and "socialized medicine" cannot be used to rally public support. Most people don't know what it is all about and care less.

On the one hand, the doctor has been pictured as one of the great men of our time with marvelous new medical and surgical treatments and techniques. On the other, he has been held up as a man very likely to lie about his income to the tax collector, a man who will not police his professional colleagues, the member of a greedy lobby, a part of the country's strongest labor union. People tend to remember that organized medicine fought against all health insurance schemes, giving in to it only when it seemed some sop must be thrown to the public to head off proponents of socialized medicine. Now, we have in Blue Cross and Blue Shield completely inadequate insurance plans for which the AMA and its affiliates take full credit.

Organized medicine must come to represent some things that are progressive and positive, not simply those things that are old and negative. To many, the straight fee-for-service method of practicing medicine is outdated and uneconomic and is bound to be supplanted. If medicine does not find a substitute, someone will do it for medicine. Modern health insurance is overpriced and inadequate, yet there are better plans available which organized medicine either ignores or damns. No matter how it may despise undeniable trends toward social living, organized medicine is doomed to a future of federal control unless it chooses to lead instead of dragging its heels. —A.B.

What Is Industrial Medicine? L. J. Wade.* Indust. Med. & Surg. 29:54 (Feb.) 1960. (*ESSO Standard Oil Co., New York.)

Rather than with "industrial medicine," we are more properly concerned with the health of people who work, whether in industry, department stores, universities, farms, or filling stations. A more proper term is "occupational medicine." The possible relationships between occupation and health or disease has been recognized for centuries. The responsibility of the employer in this area was not clearly defined prior to the workmen's compensation laws early in the 20th century. Economic pressures caused the establishment of in-plant medical services. Usually, a part-time surgeon and a nurse were retained.

Economic humanitarian and moral considerations have led most managements to seek ways to prevent injury on the job. Nonoccupational injury in the home or on the highway has become relatively a far greater hazard than injury on the job. Industrial factors leading to chronic medical disabilities were not recognized until relatively recently. These are outnumbered 100 to 1 by chronic nonoccupational diseases. Disabilities secondary to occupation account for less than 5% and in some cases 1% of all disability cases.

In-plant medical programs should consist of three activities: (1) the working environment must be under constant scrutiny for potential health hazards, (2) the worker must have assurance by medical examination from time to time that his health status is compatible with his own and his fellow workers' safety on the job, and (3) the working population must be provided with emergency medical services commensurate with the potential risk of occupational injury of illness.

Occupational medicine cannot be practiced in an office. More and more time must be spent in the study of the working environment. Prevention of illness or injury by environmental control marks an effective occupational-medical service. Clinical means of providing a safe and healthy

working environment include a medical-examination program. Dispensary services are an invaluable tool in early recognition of defects in the environmental-control program.

True industrial-medical services are oriented along the lines of preventive medicine with emphasis on the working environment and its possible relation to the health or disability of the working population. —A.B.

Radiation Health

A Limited Survey of Radiation Exposure from Medical Fluoroscopes. R. O. Gorson,* J. Lieberman, and M. Green. Radiology 73:898 (Dec.) 1959. (*Department of Radiology, Jefferson Medical College Hospital, Philadelphia 7, Pa.)

Eighty-one fluoroscopes were surveyed for radiation characteristics and manner of use by interviews with the physician, physical inspection of the fluoroscope, and radiation measurements. Fluoroscopes used by hospitals and radiologists averaged lower patient-exposure levels (8.9 r/min.) than those used by nonradiologists (16.3 r/min.). Film-badge dosimetry of physicians in private practice showed their exposure to be very low, averaging 5 mr per week for the right arm and 2 mr per week for the left shoulder.

Only two fluoroscopes met all design specifications and performance recommendations of "NBS Handbook 60." About 50% of the fluoroscopes, nevertheless, were considered not unduly hazardous. More than 90% could be made acceptable by such modifications as adding more aluminum filtration, descreasing maximum milliampere setting, decreasing maximum shutter opening, and adding accessory shielding. Improvements in fluoroscopic practice require the physician's active cooperation founded on education and understanding. Technics of flouoroscopy and the manner of use of the apparatus are not accessible to control by regulatory legislation and inspection. —G.L.V.

Environmental Radiation and Cancer. H. F. Blum.* Science 130:1545 (Dec. 4) 1959. (*National Cancer Institute, National Institutes of Health, Bethesda, Md.)

The lack of data on experimental carcinogenesis by ionizing radiation permits uncertainty regarding the shape of the curve relating radiation-dose rate and the incidence of cancer in the population. Data derived from exposure of the skin of mice to ultraviolet light, a nonionizing radiation, may promote some information if it is assumed to parallel the curve produced by ionizing radiation. Carcinogenesis by ultraviolet light is a cumulative process and, for all practical purposes, may be regarded as essentially irreversible and nonthreshold. If ionizing radiation has the same sort of relationship, the curve may be expected to have a rising inflection at low-percentage incidences of cancer; hence, a second equal increment of radiation dose should result in a greater increment of cancer incidence than that produced by the first one. In this case, estimates based on linear extrapolation would tend to minimize the effect of increase in dose rate. —G.L.V.

The Inhalation Hazard from Dust-Producing Operations in the Repair of Ships with Low-Level Radioactive Contamination. S. Goren* and J. H. Thorton, Jr. Am. Indust. Hyg. A. J. 21:71 (Feb.) 1960. (*Biological Science Division, Pacific Missile Range, Point Mugu, Calif.)

An internal radiation hazard can be encountered by personnel engaged in the repair of ships having a low-level radioactive contamination. Dust-producing operations in their apparent order of severity of hazard are disc-sanding, pneumatic wire-brushing, welding, and pneumatic chipping. The degree of hazard is dependent on the type of operation, surface-radiation intensity, distance of personnel from the operation, ventilation, and particle size. Local ventilation in the immediate area of the operation is an effective way of reducing exposures. Mechanical respirators with proper filters appear to provide adequate protection against radioactive dusts produced from contaminated surfaces up to 4.0 mrad/hr measured at 1⅝ inches from surface. In areas of higher surface contamination or when the air concentration exceeds 10^{-8} μc/cc, a clean-air-flow full-face mask is required. —G.L.V.

In Vivo Gamma Counting as a Measurement of Uranium in the Human Lung. R. E. Cofield.* Health Physics 2:269 (Feb.) 1960. (*Y-12 Plant, Union Carbide Nuclear Company, Oak Ridge, Tenn.)

Although excretion analysis is useful for the determination of uranium in the body, it cannot be used directly for evaluating that in the lungs. Therefore, an in vivo counting method was developed using scintillation spectroscopy of the gamma radiation from uranium. The equipment consists of a large sodium iodide crystal and photomultiplier, a 256-channel pulse-height analyzer, and an iron room with 8-inch-thick walls. A gamma spectrum containing peaks from the 90 kev uranium daughters, the 186 kev U^{235}, and the Cs^{137} and K^{40} contribution permits the estimation of 50% of the maximum permissible lung burden for normal, for enriched, and for depleted uranium. Careful correction for body height, weight, and shape is necessary. —T.S.E.

Radiation Protection Guidance for Federal Agencies. Federal Radiation Council* Federal Register Doc. 60-4539, May 18, 1960, p. 4402 (*1515 Massachusetts Ave. N.W., Washington 5, D. C.)

"Fundamentally, setting basic radiation protection standards involves passing judgment on the extent of the possible health hazards society is willing to accept in order to realize the known benefits of radiation." The hazards of ionizing

radiation are considered, basic biological assumptions stated, and recommendations made. The term "radiation protection guide" is used as being preferable to "maximum permissible dose." "This term is defined as the radiation dose which should not be exceeded without careful consideration of the reasons for doing so; every effort should be made to encourage the maintenance of radiation doses as far below this guide as practicable." The "guides do not differ substantially from certain other recommendations such as those made by the National Committee on Radiation Protection and Measurements, the National Academy of Sciences and the International Commission on Radiological Protection." —W.D.N.

Surgery

A Method of Tendon Repair. G. Murray.* Am. J. Surg. 99:334 (March) 1960. (*Toronto, Ont., Canada)

Murray emphasises that a repaired flexor tendon must move in its sheath before the fibrous tissue cells effecting the repair in both the tendon and the surrounding structures have time to mature and bind all structures together. If the tendon is strong enough to allow movement soon after its repair, the soft gelatinous stage of fibrous tissue cell growth will be too weak to form an attachment to the sheath and so the tendon will be left to move freely. Thus, the question of how to repair a tendon in this fashion arises.

The author outlines a method of repairing a divided tendon in an aseptic field which will withstand any tension up to normal function. He suggests the use of a segment of autogenous palmaris longus tendon at least 6 inches in length. The ends of this tendon are split into two or three strands, and the strands are interwoven through each end of the divided flexor tendon so that there is interlocking, much as in splicing a rope. The interweaving is continued back from the divided end of the tendon for not less than 2 inches. If the tendon has been divided near its terminal attachment, some of the palmaris tendon segments are passed through drill holes in the base of the terminal phalanx. The tendon sheath is left open and is sufficiently divided proximally so that the now enlarged tendon has room to permit adequate movement in finger flexion. The finger is not splinted.

The importance of continued movement following surgery is stressed. To ensure this, a nurse is instructed to sit beside the patient as he awakens from the anesthetic. She continually manipulates the finger passively in full flexion and extension. As the patient slowly regains consciousness and before pain perception returns, he is persuaded to take an active part in continuing this flexor function. Thus, the passive motion gradually becomes active. The patient is encouraged to continue this activity. If he cooperates well, a gratifying return of function is expected.

In 87% of 100 tendons repaired in this manner the author has found not less than 30 degree flexion of the distal and proximal interphalangeal joints. He has found that the average range is between 45 degrees from the distal interphalangeal joint and 60 degrees from the proximal interphalangeal joint. —J.T.A.

Treatment of Comminuted Fractures of the Calcaneus by Primary Triple Arthrodesis. K. R. Thompson* and G. M. Friesen. J. Bone & Joint Surg. 41-A: 1423 (Dec.) 1959. (*Lexington, Ky.)

The authors review their experience over a 12-year period in the management of comminuted fractures of the calcaneus by primary triple arthrodesis. Their paper considers only those fractures with displacement and involvement of the subtalar joint. They also review the various modalities of treatment dating back to the work of van Stockum in 1912, who advocated open reduction with subtalar arthrodesis.

Arthrodesis of the involved joints has been recommended by numerous investigators on the assumption that irreparable damage to the articular surfaces has occurred and that aseptic necrosis of some of the fragments with arthritic changes will develop. It is concluded that, according to the literature, comminuted, displaced calcaneus fractures cannot be successfully treated by closed manipulative procedures. The importance of special roentgenographic views is stressed by the authors. These help identify the comminuted, displaced fragments which disrupt the subtalar joint and involve the cuboid articular surface of the calcaneus at the calcaneocuboid joint. In addition, these special x-rays show the alterations of the tuber-joint angle and lateral spreading of the fragments. The severity of the fracture was found to be greater at the time of surgery than was suspected from previous review of the x-ray films. This more extensive involvement was especially noticeable in the subtalar joint. In some instances small, comminuted, detached articular fragments were found deeply imbedded in the cancellous area.

The twenty-five patients with 26 comminuted fractures of the calcaneus treated by this technique were analyzed in this study. The duration of follow-up ranged from 6 months to more than 9 years, with an average follow-up of 3.8 years. Healing time of the triple arthrodesis as judged roentgenographically by restoration of trabeculae across the arthrodesed joints averaged 3.2 months. Those patients without major associated injury returned to work on an average of six months after surgery, whereas those with associated injury required an average of eight months before returning to work. Twenty-one patients (84%) were rated as having obtained excellent results. The loss of inversion and eversion which resulted from the primary arthrodesis was not considered

by the patients to restrict any activity which they wished to perform.

The authors conclude from this study that a severe comminuted, displaced calcaneal fracture is associated with involvement of the two peritalar joints and conventional radiographic projections are not adequate for thorough examination of these fractures and two additional projections as described are necessary. They furthermore emphasize that it is their belief that the injury of the talocalcaneal joint is irreparable and that fusion of this weight-bearing joint is necessary.

—J.T.A.

Industrial Hygiene

Occupational Health Studies of the Shell-Molding Process. G. F. Tubich,* I. H. Davis, and B. D. Bloomfield. A.M.A. Arch. Indust. Health 21:424 (May) 1960. (*Michigan Department of Health, Lansing 4, Mich.)

With the introduction of shell-molding in 1950, the foundry industry experienced its first major change in operations since the Bronze Age. Since 1953 there has been a nationwide increase in the use of shell-molding, by which a mold is produced that is ⅛ to ⅜ inch in thickness. With this technique, castings have smoother surfaces, closer tolerances and thinner sections are possible, less sand is required, and production is increased.

The process utilizes a heated metal pattern over which various synthetic resin-sand mixtures are applied. The mold is placed in an oven for some 30 or 40 seconds to permit polymerization of the resin and then is removed from the pattern with the aid of stripping pins. Surface characteristics may be enhanced by the addition of chemical additives, such as various carbonates.

Environmental studies were carried out in seven Michigan foundries using the shell-molding process. Reported findings are as follows:

1. Dust: Since 80% less sand is used, it was claimed that atmospheric dust levels would be materially reduced in comparison with normal foundry operations. "The results of this study do not substantiate this claim. The data found show approximately 45% of the dust concentrations to be greater than 20 MPPCF. The high dust concentrations are due to the use of sand in a dry state in all steps of the process and lack of adequate dust-control measures." In evaluating the dust concentrations found by particle size and free silica content, it was concluded that the silicosis risk is comparable to that of a conventional foundry operation.

2. Solvents: The wide variety and types of solvents used in the process create serious problems in areas of control and explosion hazards.

3. Carbon Monoxide: Excessive levels are reported at pouring operations. Molding of shells did not cause excessive levels of carbon monoxide.

4. Resin-Decomposition Products: "The findings indicate that breathing zone concentrations of ammonia, phenol, and aldehydes, both at molding and at pouring stations, are well below their threshold limit values."

Recommendations for the control of dust and solvent-vapor are made and discussed in detail.

—L.W.S.

Some Health Hazards Associated with the Manufacture of Commercial Jet Aircraft. M. C. Robbins.* Am. Indust. Hyg. A. J. 21:182 (April) 1960. (*Aero-Space Division, Boeing Airplane Co., Seattle, Wash.)

The author divides the problems into three categories:

1. Integral wing fuel-tank cleaning and sealing.
2. Hydraulic-system testing and installation.
3. Spray painting with epoxy resin paint formulations.

The wing cleaning and fuel-tank sealing involve exposures to solvents such as methyl ethyl ketone in a confined space, plus the use of polymerizing polysulfide material with a calcium dichromate accelerator and dimethyl formamide solvent. Apparently, skin protection and local exhaust ventilation have satisfactorily solved the exposure problems and safety hazards with the exception of minor skin irritations. Careful screening on the basis of physical examination is, nevertheless, apparently necessary to rule out persons suffering from convulsive, cardiac, pulmonary, or skin diseases.

Another problem arose from hydraulic-system testing and installation where a phosphate-ester-type of synthetic fluid (Skydrol 500) is used to provide fire-resistant hydraulic fluid which will operate at temperatures from −40° to +200°F. Although preliminary patch testing indicated that this material was not a primary irritant, experience proved that more than 50% of the personnel were exhibiting varying degrees of skin reactions even among employees who were most conscientious about wearing rubber gloves. Subsequent patch testing showed that these people had become sensitized, apparently to the corrosive inhibitor in the product. As a result of subsequent tests to the products, Skydrol 7000 and Skydrol 500A have been used with less skin difficulty, although protective measures are still recommended.

Spray painting with epoxy resin paints requires not only unusually good exhaust ventilation to remove the overspray but, in addition, complete skin protection for the painters in the area during spraying operations. "Even with these precautions at least one case of sensitization has been observed among employees who have been in the area during the spraying."

—E.C.R.

Constant Flow Regulators for the High-Volume Air Sampler. W. K. Harrison, Jr., J. S. Nader, and F. S. Fugman. Am. Indust. Hyg. A. J. 21:115 (April) 1960. (*Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio.)

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This article is a rather complete analysis of the operating characteristics of the high-volume air sampler when used with and without regulators for collections of large samples over a long period of time such as is required for air-pollution sampling. Theoretical calculations and experimental tests under field conditions show a significant difference between the values of dust concentrations obtained by high-flowrate regulated and unregulated samplers. This indicates the desirability of converting unregulated samplers to the regulated type. Two regulators, operating on different principles, have been developed to maintain a constant flowrate (approximately 35 cfm) of air in a high-volume air sampler as the particulate load increases. The series regulator, being compact and simple, is immediately applicable to high-volume air samplers as an insert in the filter adapter.

—E.C.R.

Clinical Toxicology

Theory of the Causes of Silicosis—Part I. G. W. H. Schepers.* Indust. Med. & Surg. 29:326 (July) 1960. (*Haskell Laboratory, E. I. duPont de Nemours & Co., Wilmington, Del.)

This article is the first of three installments of a paper presented at the McIntyre Research Foundation Conference in January, 1960. The author includes a historical review of the recognition of silicosis as well as a comprehensive discussion of the chemical theories of causation of silicosis. The discussion includes the silica solubility theory, the sericite theory and other impurity theories, the acidity theory, the unsatisfied valency theory, and the polymeric silica theory. The final concept put forth by the author involves the changes in elemental composition of lung tissue which may occur on exposure of animals and humans to quartz dust, and the possibility that such changes may play a part in the development of silicosis. —A.D.

Theories of the Causes of Silicosis—Part II. G. W. H. Schepers. Indust. Med. & Surg. 29:359 (Aug.) 1960.

This part of the review of the theories of the causes of silicosis includes both physical and biological theories. The discussion of the physical theories includes the possible effects of particle sharpness, piezoelectricity, surface change of particles, crystallinity, particle size and surface area, solubility, and absorption properties of particles. The section on biological theories of causation includes discussions of the role of tuberculous infection, the phenomenon of phagocytosis of particles and its relationship to fibroblastic activity, the distribution of silicotic nodules in the lymphatic apparatus of the lung, the possible role of lipids in the pathogenesis of silicosis, and the effects of silica on the blood vessels of the lung. —A.D.

Renal Failure Secondary to Ethylene Glycol Intoxication. R. I. Levy. J.A.M.A. 173:1210 (July 16) 1960. (*Department of Medicine, Sinai Hospital, Baltimore, Md.)

This report describes a case of acute renal failure which terminated fatally on the 17th day of oliguria. The patient, a 36-year-old man, had a history of alcoholism for two years prior to his death and was known to have been drinking heavily during the period immediately preceding the onset of his fatal illness. No other history was available. In the course of his illness, the patient underwent dialysis twice. A presumptive diagnosis of ethylene glycol intoxication was made post mortem, when microscopic examination of the kidneys revealed many of the tubules containing bifrefringent crystals of calcium oxalate. The report includes a discussion of cases reported previously, as well as a discussion of the mechanism of toxicity involved. —A.D.

Dyspepsia Due to Inhalation of Carbon Tetrachloride Vapor. G. Kazantis* and R. R. Bomford. Lancet 1:360 (Feb. 13) 1960. (*Department for Research in Industrial Medicine, London Hospital, London E.1, England)

This report describes the effects of prolonged exposure to carbon tetrachloride vapor in concentrations of 45 to 100 parts per million on a group of workers in an electronic equipment factory. Of a total of 18 employees, histories were obtained from 17 and physical examinations were performed on 16. A large proportion of the group developed nausea and anorexia, and a significant number complained of abdominal discomfort, headache, and giddiness. Symptoms invariably increased in severity during the latter half of each working week, decreasing over the weekends. All the workers became symptom-free within one week of the control of the exposures. —A.D.

Experimental Toxicology

Production of Cancer in Mice and Rats by Chromium Compounds. W. W. Payne.* A.M.A. Arch. Indust. Health 21:539 (June) 1960. (*National Cancer Institute, Bethesda, Md.)

Sarcomas were induced in 9 of 52 mice receiving intramuscular implants of sintered calcium chromate in sheep fat and in 1 of 52 mice receiving intramuscular implants of calcium chromate, also in sheep fat; the first tumors appeared at nine months. No tumors were induced during a period of 18 months in mice injected subcutaneously with tricaprylin solutions of sintered chromium trioxide and sintered calcium chromate; one tumor in 52 mice was induced 12 months after subcutaneous injection of a tricaprylin solution of calcium chromate. The intramuscular implanation into rats of a gelatin capsule containing calcium chromate induced sarcomas in two of six after 12 months; intrapleural implantation of the capsule containing calcium chromate into rats induced sarcomas in three of

six rats. In another experiment, still in progress, no tumors have occurred in 10-12 months in rats implanted with capsules of chromic acetate.
—R.E.E.

Toxicity of Diborane in High Concentrations. A. R. Stumpe.* A.M.A. Arch. Indust. Health. 21:519 (June) 1960. (*Brooks Air Force Base, Texas.)

Toxic signs and evidence on pathology studies of severe pulmonary damage were observed in golden hamsters after inhalation of diborane. As the concentration was increased from 50 ppm (58 mg/cu m) to 600 ppm (696 mg/cu m), the mean survival time decreased from 497 to 33 minutes. Concentrations in excess of 500 ppm up to 1,000 ppm elicited no further reduction in the mean survival time. The MAC is 0.1 ppm (0.1 mg/cu m).
—R.E.E.

Intoxication by Benzene: Radioisotopes Used as Tracers. W. Paolino and L. Resegotti. Minerva med. 51:33 (Jan. 6) 1960. Abstract, J.A.M.A.

A benzene-olive oil suspension was injected intramuscularly into 18 rabbits (1 cc (400 mg)/kg every other day for four days), Fe^{59} and Cr^{51} were used as tracers. Erythrocyte counts decreased after two injections, with a marked decrease after four. Simultaneously, the animals lost weight and developed diarrhea and paralysis of the posterior limbs. There was a gradual increase in the clearance rate of Fe in the plasma after the second injection; the rate increased again after the fourth and became even more marked seven days after the last injection. The absorption rate of Fe^{59} in the hemin erythrocytes was considerably increased at the height of intoxication and during recovery; this was confirmed by blood-cell count and bone-marrow values. The life span of the red blood cells produced during the intoxication period was shorter than normal, as determined by studies using either Fe^{59} or Cr^{51} as the tagging agent. The progression rate of anemia was higher than expected.
—R.E.E.

Induction of Tumors by Tannin Extracts. K. S. Kirby.* Brit. J. Cancer 14:147 (March) 1960. (*Royal Cancer Hospital, London, England.)

Tumors were induced in August-strain rats and in stock and C_{57} black mice by weekly subcutanous injections of various tannin extracts for 12 weeks. Condensed tannins (quebracho, mimosa, and myrtan) evoked sarcomas at the site of injection as well as liver tumors. Hydrolyzable tannins (myrobalans, chestnut, and valonia) induced liver tumors only. Tannic acid had no effect when injected into rats. Mice were given, in their drinking water, 0.1% quebracho and myrtan extracts for three months and 0.5% for a further three months. No effects were noted during a period of one year. In discussing the mechanism of activity, the ability of tannins to react with proteins (forming leather) and to complex with metals (notably the complex between iron and tannic acid) and mentioned.
—R.E.E.

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The Sub-Acute and Chronic Toxicity of 1,1-Dimethylhydrazine Vapor. W. E. Rinehart,* E. Donati, and E. A. Greene. Am. Indust. Hyg. A. J. 21:207 (June) 1960. (*Army Chemical Center, Maryland.)

Exposure of mice and rats to 1,1-dimethylhydrazine at concentrations of 75 ppm for seven weeks and 140 ppm for six weeks induced tremors, convulsions, and death. In dogs, a concentration of 25 ppm for 13 weeks caused hemolytic anemia, convulsive seizure, and death. At 5 ppm for 26 weeks, only mild toxic effects were induced in dogs (slight lethargy, some hemolytic anemia, and slight bilirubinemia). The suggested tentative MAC for man is 0.5 ppm.
—R.E.E.

The Subacute Oral Toxicity to the Rat of Certain Polydimethylsiloxanes. W. E. MacDonald,* G. E. Lanier, and W. B. Deichmann. A.M.A. Arch. Indust. Health 21:514 (June) 1960. (*University of Miami, Coral Gables, Fla.)

Each of five polydimethylsiloxanes (silicones), ranging in viscosity from 50 to 60,000 centistokes, was fed to rats as 1% of the diet for 90 days. No deleterious effects were evident.
—R.E.E.

Internal Medicine

A New Flocculation Test for Differentiating Obstructive from Hepatocellular Jaundice. G. K. Daikos,* P. Mattheou and Maria Athanasiadou. Lancet 2:488 (Oct.) 1959. (*Athens University School of Medicine, Athens, Greece)

Differentiating obstructive from hepatocellular jaundice is often difficult clinically, and none of the laboratory tests have proved entirely satisfactory. The authors undertook a study to confirm Jirgl's recent findings, that the serum of patients with obstructive jaundice formed a precipitate on the addition of Folin-ciocalteau phenol reagent.

Ninety sera were examined from 29 patients with hepatocellular jaundice, 25 with obstructive jaundice and 36 with miscellaneous conditions, including hemolytic jaundice. The results confirmed Jirgl's investigation; there was flocculation in 92% of the sera when obstructive jaundice was present. No flocculation occurred in 90% of the hepatocellar cases, and there was none in the 8 cases of hemolytic jaundice. The nature of the precipitate, chemically, has not been determined; it apparently is not a mucoprotein.

The authors conclude that the test is simple to perform, and its use is recommended in the differentiation of obstructive and hepatocellular jaundice.
—S.H.S.

Cold Foot—Postoperative Sign of Silent Myocardial Infarction. N. Frank. J.A.M.A. 170:1147 (July 4) 1959.

The author describes three cases of a syndrome not previously reported; namely, postoperative

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sudden coldness of a foot in patients without known preexisting cardiac or peripheral vascular disease.

A silent myocardial infarction should be suspected if (1) a cold foot is discovered; (2) arterial pulsation are absent in the foot and/or in other palpable arteries proximally; (3) there is plantar ischemia of the foot on elevation; and (4) cardiac findings on clinical examination are normal.

It is the author's opinion that such a silent infarction occurs during anesthesia or in the immediate postoperative period. The mechanisms may be hypotension, anoxia, or cardiac arrhythmias which reduce cardiac output.

The article stresses the advisability of questioning the patient daily regarding not only pain in the foot or calf but, in addition coldness. A preoperative as well as an immediate postoperative electrocardiogram is advised. Early ambulation may prove disastrous in patients with "The cold-foot" syndrome. —S.H.S.

Diabetes and Work. E. H. W. Wijnmalen.* Diabetes 9:129 (March-April) 1960. (*Institutes of Social Medicine, Leyden University, The Netherlands)

A survey of diabetics among the employee population of three large Dutch companies was coordinated by the Institute of Social Medicine of Leyden University. A total of 464 diabetics were recognized, of whom 436 had manifested the disease after employment. The average total duration of employment was approximately 20 years, and the presence of diabetes had been known for the last 6 years. The prevalence of the disease differed in the three companies apparently because of variations in age groups. Two-thirds of those studied were more than 45 years of age.

The frequency of absenteeism was approximately the same among diabetic and nondiabetic workers, but there was some increase in the average duration. Job assignments varied among the three industries, but a number of these workers were engaged in heavy labor and some in shift work. No relationship between adequacy of job performance and the presence of diabetes was apparent. No change in job assignment or working career was necessitated by the diabetes in 70% of the cases studied. In the other 30%, changes were necessitated by complicating disease and unrelated disease.

No general statement can be made regarding the work capabilities of diabetics. Individual circumstances both with regard to the patient and the work situation are determining factors. —L.W.

The Fate of the Second Leg in the Diabetic Amputee. M. G. Goldner.* Diabetes 9:100 (March-April) 1960. (*Jewish Chronic Disease Hospital, Brooklyn, N. Y.)

Among 71 patients with "maturity-onset" diabetes and leg lesions, 12 developed such lesions to the recognition of the diabetes. The lesions are often bilateral, and the second leg of the unilateral amputee may be expected to share the fate of the first at a relatively early time. Vascular impairment in the hands was demonstrated by the method of Sigroth in 20 patients, suggesting the generalized nature of the disease. The onset of the leg lesions seemed to be more closely related to age of the patient than to the duration of the diabetes. The authors found no evidence that the severity of the diabetes or the adequacy of the control were of consequence in determining the onset of the leg lesions. —L.W.

Prognostic Features of Acute Myocardial Infarction in Men. A. E. White,* F. J. Moore, and J. Marmorston. A.M.A. Arch. Int. Med. 105:859 (June) 1960. (*Los Angeles County Hospital, Box 933, 1200 N. State Street, Los Angeles 33, California.)

Review of the hospital charts of all male patients with the diagnosis of acute myocardial infarction for the fiscal year 1956-57 at the Los Angeles County Hospital revealed 377 instances where the diagnosis was proved at autopsy, and/or the electrocardiogram was diagnostic, and/or the history was characteristic. One year follow-up studies were completed on all but 11 patients. In 22 instances the infarction occurred during the course of hospitalization for other disease. These two groups, totaling 33 patients, were discarded from further consideration, leaving 344 cases for study.

Shock on admission was associated with a very high mortality—59% during the first 24 hours, increasing to 94% at the end of a year. Age and the presence of hypertension had no apparent effect on the immediate mortality in such cases. The co-existence of failure increased the mortality appreciably. Among those patients not in shock, increased age was associated with increased mortality except in the presence of diabetes. The older diabetics seemed to fare better than the younger ones.

Arrhythmia, congestive heart failure, and hypertension were associated with an increased one-year fatality rate. Hypertension did not affect the fatality rate adversely in men beyond 70 years of age. Previous myocardial infarction had a negligible effect upon mortality. In the absence of any of the above complications, the first-year mortality rate was only about half as high. —L.W.

A New Oral Hypoglycemia Agent, Phenformin (DBI): Symposium. Alexander Marble.* Diabetes 9:163 (May-June) 1960. (*Joslin Clinic and New England Deaconess Hospital, Boston, Mass.)

The entire May-June issue of **Diabetes** is devoted to a Symposium on "A New Oral Hypoglycemic Agent, Phenformin (DBI)," held in Houston, Texas Feb. 5, 1959. The author summarizes these papers in an effort to arrive at a collective opinion regarding this new drug.

Oral or parenteral administration of DBI to most normal experimental animals results in a

lowered blood sugar level. Nondiabetic human subjects are unresponsive to DBI, but in many diabetic humans oral administration does cause a fall in blood sugar. Either endogenous or exogenous insulin seems to be necessary for a hypoglycemic effect.

No evidences of toxic effects were demonstrated in animals or humans with therapeutic doses. Numerous investigations have failed to reveal the site or mechanism of action of the drug.

The drug may be a valuable adjunct in hard to manage, unstable diabetics. It should not be used in juvenile diabetics without insulin. Some side effects, chiefly gastrointestinal, have been encountered with large doses. —L.W.

Dermatology

Intermittent Treatment of Trichophyton Rubrum Infections with Griseofulvin. M. A. Cowan.* Brit. J. Dermat. 72:185 (May) 1960. (*The Rupert Hallam Department of Dermatology, Royal Infirmary, Sheffield, England)

This practical study is aimed at comparing intermittent and continuous schedules of dosage with griseofulvin. Fifteen patients with extensive and intractable Trichophyton rubrum infections were evaluated. The patients were divided into two groups. One group received continuous treatment with griseofulvin, 2 gm daily in divided doses; the other received intermittent treatment, 2 gm daily in divided doses for two consecutive days in every seven. The drug was administered for three to nine months. Eight patients were treated by intermittent and seven by continuous dosage. There were no apparent differences in the response to treatment in the two groups. Advantages of such an intermittent dosage schedule are the smaller quantity of drug needed and the consequent lessened cost of treatment. —L.J.M.

Allergic Reaction to Griseofulvin. R. Quero.* J. Invest. Dermat. 34:283 (May) 1960. (*Havana, Cuba)

A case of an urticarial and vascular allergic eruption following ingestion of griseofulvin in a patient known to be allergic to penicillin is reported. Since both penicillin and griseofulvin are derived from the Penicillium mold, one might suspect a cross reaction between the drugs. However, as pointed out by the author, the species from which the two antibiotics are derived are quite different, and the chemical structures of the two medicaments bear no basic relationship to each other. Penicillin has a benzene ring and varied side-chains depending on the type of penicillin. Griseofulvin, contrastingly, contains a benzofuran cyclohexene nucleus. Moreover, there is much clinical experience to indicate that patients hypersensitive to penicillin are not generally hypersensitive to griseofulvin. The author therefore regards the present case as an example of a chance association of two different and distinct hypersensitivities. —L.J.M.

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Treatment of Nocardial Mycetoma with Sulphamethoxypridazine. S. Vipulyasekha* and S. Vathanabhuti. Brit. J. Dermat. 72:188 (May) 1960. (*Dermatological Clinic, Army Hospital, Bangkok)

The authors report the successful treatment of a case of nocardial mycetoma of the right foot, of 10 years' duration, with sulfamethoxypyridazine. The dose employed was 1.5 gm daily for four months. There were no untoward sidereactions. —L.J.M.

The Effects on the Skin of Some New Adhesives and Laminating Materials. G. Hodgson.* Brit. J. Dermat. 72:95 (March) 1960. (*The Royal Infirmary, Cardiff, Glamorgan, Wales)

This review is based on a paper read at a Symposium on Newer Dermatological Hazards in Industry held by the Section of Dermatology of the Royal Society of Medicine on March 19, 1959. In it the author briefly summarizes the chemistry and the effects on the skin of rubbers and synthetic resins of the formaldehyde, polyester, isocyanate, silicone, and epoxide types when used for adhesives or lamination.

NOTE: Other papers read at the Symposium reported in the same issue of the British Journal of Dermatology are "The Newer Dermatological Hazards of Industry," by S. Horner, and "The Skin Effects of Plastics," by D. K. Harris. —L.J.M.

Xylocaine (Lidocaine) Viscous. R. C. Gibbs.* A.M.A. Arch. Dermat. 81:609 (April) 1960. (*Department of Dermatology & Syphilology, New York University Postgraduate Medical School, New York City.)

Xylocaine (Lidocaine) Viscous in a 2% solution has proved to be an effective therapeutic agent in the author's 16 cases of painful oral mucous membrane lesions. —L.J.M.

Contact Dermatitis from Cinnamon. A. B. Kern.* A.M.A. Arch. Dermat. 81:599 (April) 1960. (*Department of Dermatology, Boston University School of Medicine)

Cinnamon is obtained from the bark of Cinnamomum zeylanicum but cassia (frequently used in place of cinnamon) comes from other varieties of Cinnamomum. The volatile oils of both contain as their chief ingredient cinnamic aldehyde, a powerful irritant, but, usually the amount of essential oil in the cinnamon powder employed by bakers, confectioners, etc., is very small. Since there is little mention in the literature of the etiologic significance of these substances in causing contact dermatitis, the author reports one such case in a candy maker. —L.J.M.

Intradermal Triamcinolone Acetonide in Localized Lesions. A. P. R. James.* J. Invest. Dermat. 34:175 (March) 1960. (*2140 Scottwood Ave., Toledo 2, Ohio)

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In a preliminary report, the author acquaints us with the intradermal use of triamcinolone acetonide in localized lesions. The preparation is supplied as a sterile aqueous suspension containing 10 mg of triamcinolone acetonide per cubic centimeter. The therapy is given at weekly intervals with use of a 24-gauge needle and a tuberculin syringe. James reports excellent results in 4 cases of granuloma annulare, 2 cases of sarcoidosis of the skin, 28 cases of neurodermatitis, and 2 cases of discoid lupus erythematosus. The number of injections varied from two to five. Atrophy has been the only side-effect, and it was observed in 5% of the cases. —L.J.M.

Epidemics of Tinea Capitis Due to Trichophyton Verrucosum Contracted from Cattle or Sheep. Z. Even-Paz* and F. Raubitschek. Dermatologica 120:74 (Feb.) 1960. (*Department of Dermatology and Venerology, Hadassah University Hospital, Jerusaleum)

Trichophyton verrucosum is implicated frequently in ringworm of cattle and occasionally in small epidemics of human tinea in rural areas. Two small epidemics of tinea capitis caused by T. verrucosum, affecting a total of 11 children, are described. Of significance to the industrial physician is the observation that the normal-appearing wool of some apparently healthy sheep could be pulled out easily. These hairs were shown to be parasitized. It is the contention of the authors that the use of such wool in textile manufacture might, aside from possibly resulting in weakened fabrics, lead to the infection of workers handling the raw materials. —L.J.M.

Mental Health

Oedipus, Cain and Abel, and the Geographic Full-Time System. M. Levine.* J. M. Education 35:244 (March) 1960. (*Department of Psychiatry, College of Medicine, University of Cincinnati, Cincinnati, Ohio)

Levine discusses, from the psychiatrist's viewpoint, "patterns and persons in the field of medical education and administration." He investigates some of the aggressive impulses of associate professors, assistant professors, and instructors toward the department head in a medical school. He further views the rivalry relationship between members of an academic department in such an institution.

The author concludes that the geographic full-time system—that is, the system under which faculty members give their primary interest, time, and energy to their academic appointments, are paid by the school, and are permitted to participate to a limited extent in private practice, but are not permitted to have offices for private practice outside the medical school or university hospital geographic center—provides an extremely favorable pattern for medical school departments. He feels that this is so because the system facilitates an avoidance of destructive neurotic developments in such faculty members. This concept is persented as a suggested research design, not as the final word on the subject. —A.A.M.

Morale During Personnel Cutbacks. J. T. Bair.* Personnel Administration 23:37 (July-Aug.) 1960. (*U. S. Naval School of Aviation Medicine, Pensacola, Fla.)

The author deals with the problem of sustaining employee morale during times of personnel cutback. Anticipation of job loss can lead to poor morale and attitudes of helplessness, apathy, and resignation. Occupational level and feelings of security are investigated, and Bair finds, not surprisingly, that "the higher the occupational level, the less preoccupied a man becomes with job security."

The author then discusses the relationship between anxiety and efficiency and states that this too depends upon the type of work. It is asserted that simple jobs are performed best with relatively high emotion, but "complex jobs are best performed with relatively little emotion." Is this true? Emotion is not defined but presumably refers to anxiety. Then are listed 10 recommendations based on a summary of research conducted during hard times and unemployment.

The article ends with a plea that sufficient importance should be paid to having qualified people in supervisory positions to handle people skillfully so as to sustain and develop morales. "Each supervisor must also be the personnel officer for his own people." —G.C.T.

If There's a Doctor in the House. C. P. McCord.* Supervisory Management 5:2 (July) 1960. (*Institute of Industrial Health, University of Michigan, Ann Arbor, Mich.)

In this brief article McCord presents succinctly the functions of the plant physician. One measure of the success of the medical program is the extent to which workers who require medical services do, in fact, go to the plant physician. There is, of course, the problem of the "hospital addict," and there is such a thing as laudable absenteeism.

Then, too, there is the problem of employees who have some disturbance in their emotional life. In the average plant 20% of the workers are emotionally disturbed. The author catalogues 12 danger signals which should be helpful to the supervisor or foreman. —G.C.T.

What Makes a Top Executive? D. G. Moore.* Personnel 37:8 (July-Aug.) 1960. (*Michigan State University, East Lansing, Mich.)

This article represents one of many attempts to answer the difficult question as to what characterizes the top executive. At the outset one is advised not to ask the executive himself, since he is a person of action. This might imply that the executive is not a person capable of sufficient in-

sight or reflection to answer the questions concerning his characteristics.

The author reviews briefly many of the qualities ascribed to the executive; that of being a generalist, a coordinator, a man of vast knowledge, a person who has the analytic ability to make final decisions, and, finally, a person who is simply a figurehead who participates in the ceremonial activities of the industry.

It is proposed to outline the different environments in which the executive functions rather than to list various stereotypes. These environments are taken up as follows: The value environment, the institutional environment, the company environment and the environment of occupational structure, the occupational environment, and the decision environment. Under value environment are discussed the "organization man" value and also the older value of the essential morality of work itself. Under the institutional environment, it is pointed out that industry exists along with other institutions in our society and that there is some degree of interinstitutional competition. Moore suggests that the energy and monies devoted to educational activities in a number of large corporations might better be spent by the companies on existing educational institutions. This might apply to research itself. Concerning the company environment, there are some observations about the need for employees to understand the particular way the company operates and the process whereby the individual learns the company's values, policies, procedures, and customs. Under the environment of the organizational structure, it is stated that the colleagues of the business executive and his trusted subordinates are not only his friends, the people with whom he works and cooperates in getting the job done, but they are also his enemies. They are friendly enemies; everybody smiles and carries on as if nothing really were happening, but everybody is in competition.

There is finally a section on the executive personality, listing factors such as ability to integrate, to decide, to have judgment, to be positive, and to be cooperative. —G.C.T.

A Poet Speaks to Businessmen. B. Mills.* Harvard Business Rev. 38:103 (Sept.-Oct.) 1960. (*Department of English, Purdue University, Lafayette, Ind.)

Since occupational medicine operates within the framework of industry, it quite understandably tends to accept the philosophy of the business man or at least combine it with that of a physician. The chief values are economic and technological, together with the manipulation of power. All are intensified by mass communication.

This article presents another point of view, that of the poet, and in so doing introduces some refreshing opinions. The poet has always rejected the notion that money, status, cash balances, referendums, and production curves are the real measure of living. He considers them peripheral

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or even antithetical to the essential human experience. He deplores the dead use of language in the cliché, the stereotyped phrase, jargon, and all kinds of gobbledygook found in business, government, education, and even in most talk about poetry. Dishonest word usage is double talk —the half-truths of propaganda, advertising, slick politicians, and the hard sell. —G.C.T.

Noise and Hearing

Middle Ear Muscle Activity at Moderate Sound Levels. F. B. Simmons.* Ann. Otol. Rhin. & Laryng. 68:1126 (Dec.) 1959. (*Department of Neurophysiology, Walter Reed Army Institute of Research, Washington, D. C.)

The function of the middle-ear muscles (stapedius and tensor tympani) in the transmission of sounds of moderate intensity was studied in cats. By means of electrodes permanently implanted on the round window, the cochlear microphonic response (CM) to a tone presented to the ear by means of a hearing-aid receiver could be measured in ears with one, both, or neither of the muscles severed, the cats being either anesthetized or awake. If both muscles were cut, the measured CM was the same whether the cat was awake or anesthetized; but, if the muscles were intact, then the magnitude of CM was much smaller in the awake condition than under anesthesia. This reduction in amplitude, representing the sound attenuation due to the muscles, was greater for frequencies below 2000 cps than above and was nearly as great for moderate intensities (about 40 db above 0.0002 dynes/cm^2) as for high intensities (95 db); the attenuation amounted to about 10 db at the low frequencies. Study of ears with only one muscle or the other intact showed the stapedius to be mainly responsible for this attenuation, a finding that raises some questions as to the meaning of studies that have employed the movement of the tympanum (which is largely controlled by the tensor tympani) as indicating the action of the protective reflex.

—W.D.W.

Protective Effect of the Acoustic Reflex for Impulsive Noises. J. L. Fletcher* and A. J. Riopelle. J. Acoust. Soc. America 32:401 (March) 1960. (*U.S. Army Medical Research Laboratory, Fort Knox, Ky.)

Twenty-four men were exposed twice to the noise generated by 100 rounds of .30-caliber gunfire fired semirandomly over a period of seven minutes. In one case each shot was preceded by a 1000-cps tone at 98 db SPL (sound pressure level) which activated the auditory protective reflex; in the other exposure, this activating tone was omitted. In the latter case, the auditory reflex presumably acts too late to afford any protection. The peak level of the acoustic pulses produced by this gunfire is said to be above 132 db SPL. When

the activating tone was used, the average temporary threshold shift at 6000 cps (the frequency showing the greatest effect) was 13 db, while omission of the activating tone resulted in a 23-db shift. This difference of about 10 db in the shifts produced with and without the activating tone was found for other test frequencies as well. Thus, the value of the auditory reflex in attenuating impulsive as well as steady sounds is demonstrated. —W.D.W.

The Intratympanic Muscle Reflex as a Protective Mechanism Against Loud Impulsive Noise. D. A. Hilding.* Ann. Otol. Rhin. & Laryng. 69:51 (March) 1960. (5639A Folger St., Fort Knox, Ky.)

This experiment is an extension of the results of Fletcher and Riopelle on the diminution of temporary threshold shifts from gunfire produced by prearousal of the auditory reflex to the protection against permanent losses afforded by the reflex. Electrodes were permanently implanted at the round windows of 10 cats, and the cochlear microphonics produced by test tones were measured before and after exposure to 2,000 rounds of .30-caliber gunfire presented at intervals of two to six seconds (the peak SPL is estimated to be 155-160 db). For five of the cats, each shot was preceded by the reflex-arousing tone; for the other five, the tone was omitted. The stapedius tendon had been severed in some ears and the tensor tympani in others. Profound losses were produced in all 10 ears exposed without the arousal tone and, of the cats exposed with arousal tone, in the 3 ears with severed stapedius tendons. Ears with severed tensor tympani or with both tendons intact showed only negligible changes after exposure with arousal tone. It is clear that prearousal of the auditory reflex does afford protection against damage from impulsive sounds and that most of the protection can be attributed to the stapedius. However, the tensor tympani gives some attenuation: the 3 ears with intact tensor tympani referred to above showed less damage than the 10 ears exposed without arousal tone.

—W.D.W.

Masking of Speech by Bands of Noise. I. J. Hirsh* and W. D. Bowman. J. Acoust, Soc. America 25:1175 (Nov.) 1953. (*Central Institute for the Deaf, St. Louis, Mo.)

This experiment was designed to measure the shift in the threshold of intelligibility of spondee words in the presence of various bands of noise as a function of the over all level of the noise band. Spondee words were presented in the quiet and in the presence of different levels of bands of noise, each band width corresponding to a pitch interval of 250 mels plus one white noise band. The absolute thresholds for these bands were also determined to permit transformation of the noise level from an absolute scale to a scale of sensation level. The results indicate linear relations between the masked threshold of speech and the level of the masking noise for white noise and for bands between 394 and 1420 cps. This masking function is accelerated for the noise bands above 1420 cps, while the two bands below show first positive and then negative acceleration. The most effective band to mask speech contains frequencies between 670 and 1000 cps. Less masking is provided at the same levels at either higher or lower frequencies. When the absolute thresholds for the noise bands are converted to sound-pressure level per critical band, they agree well with the absolute thresholds for pure tones at the center of these bands. The signal to noise ratio for the threshold of intelligibility for spondee words heard in white noise is –15 db, for the middle frequency bands it is –25 db, and for the highest and lowest bands it is as low as –60 db. —R.G.H.

Survey of Hearing in a Low Industrial Noise-Level Population. J. F. Corso.* WADC Technical Report 58-442, Wright Air Development Center, October, 1958. (*The Pennsylvania State University, University Park, Pa.)

The purpose of this survey was to obtain data on the hearing characteristics of a large number of subjects drawn from a population exposed to minimal levels of industrial noise. This information might help resolve some problems, such as the establishment of a "baseline" or "normal" threshold of auditory sensitivity for both pure tones and speech. Threshold data from different age groups would also be of value in evaluating the effects of presbyacousia with little confounding due to high intensity noise effects. Thus, these data could provide a set of reliable threshold norms which might be utilized in subsequent medicolegal cases involving the assessment of hearing loss due to excessive exposure to high intensity noise.

Nine different studies are reported in detail, including the procedures and tests used along with the results and conclusions of each study. These studies include the threshold of hearing for pure tones as measured by different audiometric techniques, variables of the operating conditions using Békésy-type audiometers, and a clinical discrete frequency audiometer; the method of adjustment and method of limits are reported. The effects of testing methods on hearing thresholds were also studied. The intersubject and intrasubject variability was compared, and the effects of practice on absolute auditory thresholds were defined. The threshold of hearing for speech and the discrimination loss for speech were studied. The results of this extensive hearing study are reported in terms of normal threshold values, and they provide an adequate basis from which hearing loss due to noise exposure may be evaluated. The author recommends that the present standards of normal hearing be reestablished to account for the factors of age and of sex as important determinants of hearing ability. —R.G.H.

About the Authors

► JOSEPHINE A. CIPOLLA, R.N., M.P.H., is senior author of our lead article in this issue, "Occupational Health Service Experiences of Two Hotels for 1955 and 1956." The article is based on a study performed by her in connection with graduate work at Yale University.

Born in Italy, Miss Cipolla received her education in this country. She took nurse's training at Deaconess Hospital, Buffalo, N.Y., and attended Canisius College in that city. She later enrolled at Columbia University Teachers College in New York City and received the B.S. degree from that institution. She studied in the industrial nursing program at Yale, and was awarded the M.P.H. degree in 1958.

Miss Cipolla has worked as a general duty nurse and emergency room nurse in Buffalo hospitals and has had an extensive experience as an industrial nurse with Hilton Hotels Corporation. She is currently on the administration staff of the American Nurses Association in New York City.

► J. WISTER MEIGS, M.D., is Associate Professor of Occupational Medicine at Yale University, the Director of Employee Health Service at Yale, and Works Physician (New Haven Works) to American Steel and Wire Division, United States Steel Corporation.

Dr. Meigs received the A.B. degree from Princeton University, Princeton, N. J., and the M.D. degree from Harvard University, Boston, Mass. He interned at the Pennsylvania Hospital, Philadelphia, for 18 months during 1940-1942 and then took a residency at the Massachusetts General Hospital, Boston. After a short period as a medical officer of Alaskan Division, Pan American Airways, Seattle, Wash., Dr. Meigs became Chief, Medical Section, Industrial Hygiene Service, U.S. Army. He joined the Yale faculty in his present capacity in 1947.

Dr. Meigs is widely known for his many activities in industrial medicine. He serves on local and national committees on rehabilitation and is Consultant in Occupational Health to the Surgeon General, U.S. Army. His publications reflect his own research work in the toxicity of benzidine, carbon monoxide, and other industrial substances. He has for some time worked at the solution of the problem of occupational health services for small industries.

Dr. Meigs is certified in occupational medicine by the American Board of Preventive Medicine. He is a fellow of the American Academy of Occupational Medicine and of the Industrial Medical Association. He is co-author of the article in this issue, "Occupational Health Service Experiences of Two Hotels for 1955 and 1956."

► MARY LOUISE BROWN, R.N., M.A., is a leading figure in nursing education circles. As Assistant Professor of Public Health (Occupational Health Nursing) at Yale University School of Medicine, she directs the only current university program of graduate training for industrial nurses.

Miss Brown took nurse's training at Western Pennsylvania Hospital, Pittsburgh, Pa., where she received the R.N. in 1938. Thereafter she was successively Student Supervisor at Western Pennsylvania Hospital; an industrial nurse at Duquesne Works, U.S. Steel Corporation; and an instructor in psychiatric and industrial nursing at the University of Pittsburgh. During this period she continued studies at Pittsburgh and received the B.S. degree (with honors) in Nursing Education, and an M.A. in mental health nursing. She was elected to Sigma Theta Tau, national honorary society for nursing.

Miss Brown is a member of the American Nurses Association, AAIN, and the American Public Health Association. She is a member of the board of directors of the National League for Nursing, and is president of the Connecticut unit of that organization. She is the author, with J. Wister Meigs, M.D., of the textbook "Occupational Health Nursing" (Springer Publishing Company, New York, 1956).

► FRANK P. GUIDOTTI, M.D., has been the recognized medical leader in the development of organized medical care for the hotel workers of New York. He entered this activity when it was at the planning stage early in 1950, and shortly thereafter became Medical Director, New York Hotel Trades Council and Hotel Association of New York City Health Center, Inc. As suggested by the organization's long name, this is a cooperative union-management enterprise.

Dr. Guidotti, a native of New Jersey, had his premedical education at Bucknell University. He was enrolled at Jefferson Medical College in Philadelphia during 1929-1931 and completed his medical education at the Medical School of the Royal University of Naples over the next two years. After an internship at the Jersey City Medical Center, he developed a general practice that included industrial surgery for various business concerns in and around Trenton. He was, in addition, a contract physician for examination of recruits for the U.S. Army, an activity that led directly into an unusual military experience.

Dr. Guidotti volunteered for military service in October, 1940, and was in active service until April, 1947. He planned, organized, and directed the medical services at Trenton, Fort Jay, and Grand Central Palace (New York); became the Surgeon of Recruiting and Induction of Second Service Command; and organized and supervised the medical services in connection with Army Ground and Service Forces Redistribution Stations at Lake Placid and at Atlantic City, and later at the separation centers at Fort Dix and Fort Monmouth. He achieved the active rank of lieutenant colonel.

At the conclusion of his war service, Dr. Guidotti began a practice in New York City, in which he provided medical examination services for a number of firms and institutions. These activities led directly into his present full-time employment as Medical Director of the Hotel Health Center. He is the author of the article in this issue, "Medical Care and Welfare Program for Hotel Workers in New York City."

About the Authors

► CAPTAIN HARRY J. ALVIS, a graduate of the University of Iowa College of Medicine (1933), was engaged in private practice in Orgeon when mobilized as a reserve naval medical officer in January 1941. His service career has included duty with the Marines, aboard a battleship, at Oakland Naval Hospital, as a medical department instructor for ships being placed in commission, and in medical intelligence work in Europe. His interest in medical problems of the workman and his work place, stimulated by the problems of the logging industry in Oregon, were translated into the naval situation by the challenges facing the submarine service. While in Europe (1945-1948) he fostered the compilation of German research in the field of submarine operations and also began the work still continuing in the Atlas of Epidemiology. Upon return to the United States, he attended Harvard School of Public Health (1949) and has been associated with the problems of submarines and deep-sea diving ever since. He has served the submarine forces in various capacities, including Force Medical Officer, Submarine Forces, Atlantic Fleet; Director, Submarine Medicine Division, Bureau of Medicine and Surgery; and, currently, Force Medical Officer, Submarine Force, Pacific Fleet. Dr. Alvis is certified in Occupational Medicine by the American Board of Preventive Medicine. He is the author of "How Deep is Dangerous?" in this issue.

► BURTON C. KILBOURNE is senior author of a series of reviews beginning in this issue: "The Treatment of Industrial Hand Injuries: I. General Principles of Wound Treatment and Injuries to Enveloping Tissues." This is his eleventh journal publication on injury treatment. He is a co-author, with Eudell G. Paul, M.D., of "First Aid in Industry" (Appleton-Century-Crofts), now in its fifth edition.

After receiving the M.D. degree from Northwestern University and completing an internship in St. Luke's Hospital, Chicago (1936), Dr. Kilbourne embarked on a career that combined service with International Harvester Company and progress in the field of surgery. He practiced for two years at the Harvester Company's mine community at Benham, Ky., and then transferred to Milwaukee, Wis. At the latter place he served as a voluntary instructor in anatomy at Marquette University. He then went to Chicago, where he continued his connection with Harvester; began a private practice in surgery; and assumed teaching responsibilities, at the University of Illinois and later at Northwestern University.

Dr. Kilbourne was Chairman of the Department of Surgery at St. Luke's Hospital during the years 1957-1959, and is now Attending Surgeon at the combined Presbyterian-St. Lukie's Hospital. He is Clinical Associate Professor of Surgery at the University of Illinois. He has been Assistant Director of Medical Services at Harvester since 1941. Dr. Kilbourne is a Fellow of IMA. He is a diplomate of the American Board of Surgery, and is a member of Alpha Omega Alpha honorary fraternity.

► EUDELL G. PAUL, M.D., attended the University of Illinois, at Champaign-Urbana, Ill. He received the M.D. degree from George Washington University School of Medicine, Washington, D.C. After an internship at St. Luke's Hospital, Chicago in 1946-1947, he served a four-year residency in that hospital. He became a diplomate of the American Board of Surgery in 1956.

Dr. Paul is on the medical staff of International Harvester Company, and is responsible for the surgical care of that firm's Wisconsin Steel Company employees at South Chicago Community Hospital. In addition he carries on a private practice and is a clinical assistant in surgery at the University of Illinois. He is Assistant Attending Surgeon at Presbyterian-St. Luke's Hospital.

Dr. Paul is a member of IMA and the American Medical Association. He is co-author of the surgical review in this issue: "The Treatment of Industrial Hand Injuries: I. General Principles of Wound Treatment and Injuries to Enveloping Tissues." —EDITOR

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People and Events

► The creation of a new Division of Occupational Health within the U.S. Public Health Service has been announced by the Surgeon General. The new Division, which replaces the Occupational Health Program, is designed to strengthen the PHS's program to protect the health of American workers. It places greater emphasis on all types of environmental health hazards and provides for "long-needed increased efforts" in the occupational health field as recommended by the Study Group on Mission and Organization of the Public Health Service.

HAROLD J. MAGNUSON, M.D., has been designated Chief of the new Division of Occupational Health. He has directed the occupational health activities of the Public Health Service for the past four years and previously had served as Chief of Operational Research for PHS's venereal disease program.

To keep pace with a changing technology and multiplying hazards in the work environment, the Division will carry out an expanded research program directed at developing better techniques, materials, and equipment for use in the prevention, diagnosis, and treatment of occupational disease. Major research efforts will be intensified in toxicology and in related fields of clinical medicine, engineering, chemistry, and physics. In addition, physiological and psychological factors in the work environment will come under more penetrating study.

Greater emphasis will also be focused on the training of personnel in occupational medicine and nursing and in industrial hygiene to meet the needs of this rapidly growing field. The Division will work with academic institutions and other groups to help develop the necessary professional resources.

A third area of major expansion is in state aid services, designed to support and assist official agencies in the development and operation of occupational health programs throughout the nation. The Division will also be responsible for stimulating effective community and industrial efforts to supply occupational health services.

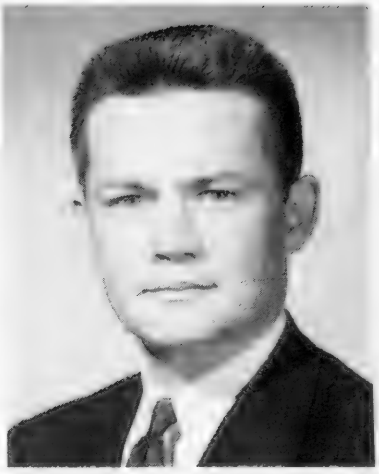
Research and technical services, as well as training activities, will be carried out at Cincinnati, Ohio. State aid activities, occupational health nursing consultation and studies and consultation relating to employee health programs will be centered at the Division's administrative headquarters in Washington, D.C.

Harold J. Magnuson, M.D.

DR. MAGNUSON received his M.D. degree in 1938 from the University of Southern California and the degree of Master of Public Health in 1942 from the Johns Hopkins School of Hygiene and Public Health. He was commissioned in the Public Health Service in 1941.

DR. MAGNUSON is certified by the American Board of Preventive Medicine and Public Health. He is a member of the American Medical Association, the Society of American Bacteriologists, the American Society for Clinical Investigation, the American Society of Experimental Pathology, and the Industrial Medical Association. He is also a Fellow of the American College of Physicians, the American Public Health Association, and the American Association for the Advancement of Science.

► GILBERT B. MEYERS, M.D., has been appointed Medical Director of Socony Mobil Oil Company's installations at Paulsboro, N.J., succeeding CHESTER L. SAMUELSON, M.D., who now heads the Ohio Oil Company's medical department.



Gilbert B. Meyers, M.D.

DR. MEYERS attended Carnegie Institute of Technology and is a graduate of St. Vincent College, Latrobe, Pa. He received his M.D. degree from the University of Pittsburgh School of Medicine in 1947 and served his internship at Mercy Hospital in Pittsburgh. In 1948, he joined the medical department of Curtis Publishing Company, where he remained until 1954. He was named manager of health services at the Radio Corporation of America's Camden, N.J., plant in 1955, a position he held until his present appointment.

DR. MEYERS is an assistant professor in public health and preventive medicine at Hahnemann Medical College, Philadelphia. He is a Fellow of the Industrial Medical Association, and a member of the American Medical Association, American Academy of Occupational Medicine, American Industrial Hygiene Association and the Medical Club of Philadelphia.

► EDWIN A. MCGOVERN, M.D., has been appointed Works Physician at the Pittsburgh Works of Jones & Laughlin Steel Corporation. He fills a position left vacant when ROBERT J. HALEN, M.D., was named Medical Director for the company in June of this year.

DR. MCGOVERN has been Works Physician at J & L's Cleveland Works since 1953. He received his bachelor's degree from Providence

People and Events

College, Providence, R.I., in 1942 and his M.D. degree from Jefferson Medical College in 1946. He later earned a Master of Public Health Degree at the University of Pittsburgh's School of Public Health.

Before joining Jones & Laughlin, DR. MCGOVERN was Plant Physician at the General Motors Cadillac Tank plant in Cleveland. Prior to that time he was a Resident Physician in Internal Medicine at the Veterans Administration Hospital at Aspinwall, Pa. He served in the U.S. Medical Corps from 1947 to 1949.

DR. MCGOVERN is a Diplomate of the American Board of Preventive Medicine in Occupational Medicine: and a member of the American Medical Association, Industrial Medical Association, Cleveland Academy of Medicine, and the Medical Directors' Club of Cleveland.

Edwin A. McGovern, M.D.

► CAPT. LLOYD B. SHONE (MC), USN, retired from the Navy on August 1, following more than 30 years of service. At the time of his retirement he was Director of the Occupational Medicine and Dispensary Division of the Bureau of Medicine and Surgery.

REAR ADMIRAL B. W. HOGAN, Surgeon General of the Navy, honored CAPTAIN SHONE on the occasion of his retirement by presenting him with the Bureau of Medicine and Surgery Certificate of Merit for his outstanding service. The presentation was made in a ceremony before a gathering of friends and fellow officers.

CAPTAIN SHONE'S navy career began during World War I when he was an enlisted pharmacist's mate. He is one of the few Navy doctors who holds both the Doctor of Medicine and the Doctor of Dentistry degrees. He earned his D.D.M. degree at Harvard Dental School in 1923, and his M.D. degree from Creighton University in 1933. Shortly thereafter he was commissioned a Lieutenant in the Medical Corps of the U.S. Naval Reserve. He has served continuously on active duty since 1940, and attained the rank of Captain, Medical Corps, U.S. Navy in 1945. He holds the following medals and awards: American Defense Service Medal; American Area Medal; European-African-Middle East Area Medal; Asiatic Pacific Area Medal; National Defense Service Medal; and World War I and World War II Victory Medals.

CAPTAIN SHONE is a Fellow of the Industrial Medical Association and a member of its Nominating Committee. He is a Fellow of the American College of Preventive Medicine, and the American Public Health Association and is a member of the American Academy of Occupational Medicine, the American Medical Association and the Association of Military Surgeons of the United States.

He will make his home in San Francisco where he will continue to be active in the field of occupational medicine.

Rear Admiral B. W. Hogan (right) presenting a "Certificate of Merit" to Capt. Lloyd B. Shone of the Medical Department of the U. S. Navy.

► LCDR NORBERT E. ROSENWINKEL (MC), USN, has been appointed Director of the Occupational Medicine and Dispensary Division of the Bureau of Medicine and Surgery, Navy Department, to fill the vacancy occasioned by the retirement of CAPT. LLOYD B. SHONE.

During World War II, COMMANDER ROSENWINKEL served as an enlisted man in the medical department of the U.S. Army. Immediately after the war, he entered the Medical School of Louisiana State University graduating with an M.D. degree in 1949. He began his active duty as a commissioned officer in the Medical Corps of U.S. Navy in July, 1949. He is qualified in both aviation and occupational medicine, and was designated a flight surgeon in 1951. In 1958 he received the degree of Doctor of Public Health from the University of Pittsburgh.

COMMANDER ROSENWINKEL'S professional affiliations include: American Medical Association; Industrial Medical Association; and the Association of Military Surgeons. He is a Diplomate of the American Board of Preventive Medicine, certified in the specialty of Occupational Medicine.

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People and Events

► R. LOMAX WELLS, M.D., has been reappointed Chairman of the Medical Advisory Council, American Association of Industrial Nurses, it was announced by MARGARET L. STEELE, R.N., AAIN president.

The Medical Advisory Council, which DR. WELLS has headed since 1959, includes the following industrial physicians: D. J. LAUER, M.D.; GLENN S. EVERTS, M.D.; JAMES P. HUGHES, M.D.; O. T. MALLERY, JR., M.D.; D. D. HOLADAY, M.D.; RICHARD C. SWAN, M.D.; B. DIXON HOLLAND, M.D., and LOGAN T. ROBERTSON, M.D. This group counsels with the national industrial nursing group on matters relating to the practice and principles of industrial medicine and nursing.

DR. WELLS is the Medical Director of the Chesapeake and Potomac Telephone Companies, with headquarters in Washington, D.C. He is a Fellow and past member of the Board of Directors of the Industrial Medical Association; a Fellow of the American College of Physicians, the American Academy of Occupational Medicine, and the American College of Preventive Medicine; a Diplomate of the American Board of Preventive Medicine (Occupational Medicine); a member and past chairman of the Section on Industrial Medicine, Southern Medical Association; and Past Vice-President and Chairman, Committee on Industrial Health, District of Columbia Medical Society.

R. Lomax Wells, M.D.

Miles O. Colwell, M.D.

► MILES O. COLWELL, M.D., has been named Medical Director for the Aluminum Company of America, Pittsburgh, succeeding DUDLEY A. IRWIN, M.D., who has retired. DR. IRWIN organized Alcoa's medical and industrial hygiene program in 1943.

DR. COLWELL began doing part-time work for Alcoa in 1952 while he was engaged in the private practice of internal medicine. He joined the medical department on a full-time basis in March, 1959, as Medical Director of the New Kensington plant and research laboratory. Later he became Assistant Medical Director of the Company.

DR. COLWELL received his M.D. degree from the University of Pennsylvania in 1943 and served his internship at Presbyterian Hospital in Philadelphia. From 1944 to 1946 he served as an Army battalion surgeon and was awarded the combat medical badge, Bronze Star and Silver Star. He returned to Presbyterian Hospital for his residency in internal medicine in 1946. He is a member of the Industrial Medical Association, the American Medical Association and the American Diabetes Society and is a Diplomate of the American Board of Internal Medicine.

► JOHN MACIVER, M.D., has been appointed Assistant Medical Director of the United States Steel Corporation. He will be associated with ROBERT B. O'CONNOR, M.D., Medical Director, in the Company's headquarters offices in Pittsburgh.

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DR. MACIVER was occupational psychiatrist for the American Fore Loyalty Group in New York City. Prior to that he was Director of the Safety and Occupational Health Bureau, Metropolitan Life Insurance Company.

DR. MACIVER graduated from Rutgers University in 1944 and obtained his M.D. degree from the College of Physicians and Surgeons of Columbia University in 1949. He did post-graduate work in the Department of Psychiatry at Yale University and earned the degree of Master of Public Health in 1953.

He is a member of the Industrial Medical Association, the American Psychiatric Association and the American Public Health Association. He is a Diplomate of the American Board of Preventive Medicine, certified in the specialty of Public Health.

► WILLIAM J. BURNS, Executive Director of the Michigan State Medical Society, and Director of Exhibits for the Industrial Health Conference, was honored recently by the American Society of Association Executives at its 41st annual conference held in French Lick, Ind.

MR. BURNS received the "Key Award" for the outstanding association executive in the state association category. His citation, presented along with the plaque, included a lengthy list of "firsts": first Volunteer Secretary of Michigan Medical Service, first Secretary of Michigan Health Council, first Secretary of Michigan Foundation for Medical and Health Education, first Secretary of Michigan Cancer Coordinating Committee, first Secretary of the Organizing Committee of the Michigan Heart Association and first Secretary of the Beaumont Memorial Foundation. In addition, Mr. Burns was active in the formation of the Michigan Association of the Professions, and of the Association Forum of the Association Executives of Michigan. He has served as Executive Director of the Michigan State Medical Society since 1936.

► Hawaii Association of Plantation Physicians will hold its annual meeting October 6 through 9 at Agee Hall, Experiment Station, Hawaiian Sugar Planters' Association, Honolulu.

A panel discussion on "Urological Problems in the Aging and How to Treat or Prevent Them" will be conducted under the chairmanship of MARVIN BRENNECKE, M.D., with ELMER HESS, M.D., as guest speaker. Panel members will include ANDREW MORGAN, M.D., EDMUND ING, M.D., and T. D. WOO, M.D.

RICHARD NODA, M.D., will conduct another panel discussion on "Geriatrics, Our Increasing Problem." Other presentations on the program include: "Carotid Thrombosis Simulating Stroke and Its Surgical Correction"—RALPH CLOWARD, M.D.; "Hypertension"—MORTON BERK, M.D.; "Chronic itch"—CYRUS LOO, M.D.; "Use of Isotopes"—GEORGE BURR, M.D.; "Toxicity Materials in Use on Plantations"—KENNETH SUND, M.D.; and "The Medical Profession and Data Processing"—GEORGE DARROCH.

The Program at the banquet will feature brief presentations of "Old time" plantation medicine by JAY KUHNS, M.D., L. L. SEXTON, M.D., C. L. CARTER, M.D., and A. L. DAVIS, M.D.

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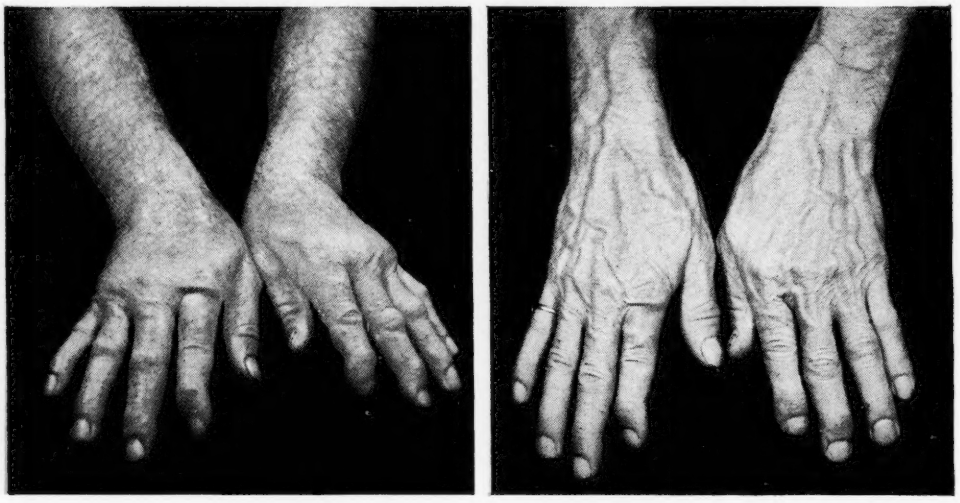

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► Quebec and Ontario Industrial Medical Conference will be held October 12, 13 and 14 at Britannia Lodge near Huntsville, Ontario. The meeting is co-sponsored by the Quebec Industrial Medical Association and the Section on Industrial Hygiene of the Ontario Medical Association.

The program includes the following presentation: "Clinical Demonstration of Examination of the BACK"—DR. G. F. PENNAL; "Some Common Aspects of the Pneumoconioses"—DR. K. W. SMITH; "Medical Research in the Petroleum Industry"—H. W. GERARDE; "By Hand or Mouth?"—DR. D. K. GRANT; "Infectious Hepatitis"—DR. J. C. SINCLAIR; "Newer Trends in the Treatment of Tuberculosis"—DR. J. F. PATERSON; "Some Physiological Effects of Exposure to Cold"—DR. J. HUNTER; "Radiation Hazards and Their Control"—DR. G. BUTLER; "Clinical Effects of Ionizing Radiation"—DR. G. STEWART; "Application of Ionizing Radiation to Industry with Control Measures"—DR. L. B. LEPPARD; "An Approach to Alcoholism in Industry"—DR. J. H. BAILLIE; "Traffic Accidents and Their Emergency Care"—panel discussion including DR. A. MCKILLOP, chairman, C. LAYBOURN, DR. R. M. MUSTARD and DR. A. W. PEART.

► New England Industrial Medical Association will hold its annual meeting at the Hotel Somerset, Boston, on November 2. VALMORE A. PELLETIER, M.D., Chief of Surgery, Norwood Hospital, Boston, will speak on "The Diagnosis of Abdominal Pain." A business meeting and election of officers and directors will follow.

► The Central States Society of Industrial Medicine and Surgery will hold its winter meeting at the Milwaukee Inn, Milwaukee, Wis., on December 3.

► Western New York Society of Industrial Medicine and Surgery will hold an all-day meeting October 22 at the Treadway Inn in Niagara Falls. Two papers which will be presented in the morning are "Three-Year Myocardial Infarction Study in the Dupont Company"—C. A. D'ALONZO, M.D., Assistant Medical Director, E. I. du Pont de Nemours & Co., Inc.; and "Arthritis in Industry"—SALVATORE LATONA, M.D., Chief of Medicine, Mt. St. Mary's Hospital, Niagara Falls, and Instructor of Medicine, University of Buffalo School of Medicine. The afternoon program will consist of a tour of the Niagara Power Project.

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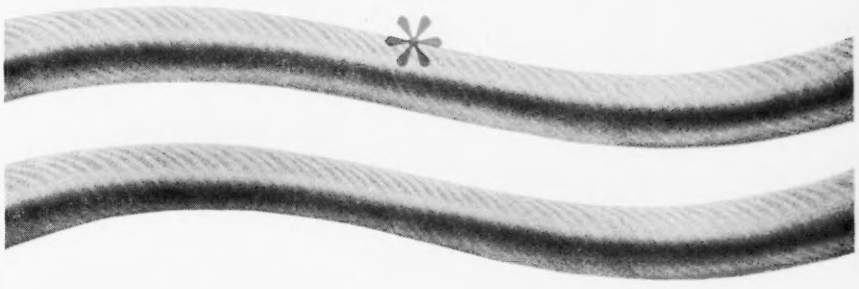

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